



ACTA ORTHOPAEDICA SCANDINAVICA  
SUPPLEMENTUM no 49

---

FROM THE ORTHOPAEDIC CLINIC LUND  
CHIEF PROFESSOR C WIBERG

COMPARATIVE VALUE OF  
ELECTROMYOGRAPHIC, MYELOGRAPHIC  
AND CLINICAL-NEUROLOGICAL  
EXAMINATIONS IN DIAGNOSIS OF  
LUMBAR ROOT COMPRESSION SYNDROME

BY

BERTIL KNÜTSSON



EJNAR MUNKSGAARD  
COPENHAGEN 1961





COMPARATIVE VALUE OF  
ELECTROMYOGRAPHIC MYELOGRAPHIC  
AND CLINICAL NEUROLOGICAL  
EXAMINATIONS IN DIAGNOSIS OF  
LUMBAR ROOT COMPRESSION SYNDROME





FROM THE ORTHOPAEDIC CLINIC LUND  
CHIEF PROFESSOR C. WIBERG

COMPARATIVE VALUE OF  
ELECTROMYOGRAPHIC, MYELOGRAPHIC  
AND CLINICAL-NEUROLOGICAL  
EXAMINATIONS IN DIAGNOSIS OF  
LUMBAR ROOT COMPRESSION SYNDROME

BY

BERTIL KNUTSSON



IJNAR MUNKSGAARD  
COPENHAGEN 1961



# CONTENTS

|             |  |     |
|-------------|--|-----|
| Chapter I   | <i>Introduction</i>  | 7   |
| Chapter II  | <i>Historical review</i>   | 8   |
| Chapter III | <i>Material</i>  | 18  |
|             | Classification   | 18  |
|             | Projection of pain   | 23  |
|             | Clinical neurological examination  | 28  |
|             | Changes in the Achilles tendon reflex  | 28  |
|             | Changes in the patellar reflex   | 34  |
|             | Weakness or paralysis in the ext of the great toe<br>(+paresis and atrophy of musc ext dig brev) | 38  |
|             | Impaired sensibility   | 42  |
|             | Muscular atrophy   | 46  |
|             | Lasegue's test   | 48  |
|             | Myelographic findings  | 48  |
|             | Electromyographic findings   | 61  |
| Chapter IV  | <i>Correlation of different diagnostic methods with operative findings</i>                       | 71  |
| Chapter V   | <i>The review</i>  | 92  |
|             | Results  | 92  |
|             | Clinical findings  | 95  |
|             | Electromyographic findings   | 99  |
| Chapter VI  | <i>Discussion and Conclusions</i>  | 101 |
| Chapter VII | <i>Summary</i>   | 119 |
|             | <i>References</i>  | 123 |



## INTRODUCTION

Occasionally clinical neurological as well as myelographic examination will fail to indicate the level of lumbar root compression. There is, however, a third method which has not been widely used for this purpose, namely, electromyography.

After perusal of the literature on the clinical application of electromyography in lumbar nerve root compression syndrome, the present investigation was started. The purposes of which were to find answers to the following questions:

- 1) To what extent can electromyography be utilized in the investigation of the level of lumbar root compression syndrome?

- 2) To what extent do denervation potentials persist post-operatively? (Can the method be used for recurrences?)

In the investigation patients with sciatica were examined:

- 1) electromyographically by the author personally who had no knowledge of the patient's history or clinical findings

- 2) clinically neurologically by the author personally

- 3) myelographically (Abrodil) at the Roentgen Department II University Hospital I and

- 4) electromyographically and clinically neurologically 1 year after the operation

## HISTORICAL REVIEW

Sciatica — formerly a syndrome of unknown aetiology — has aroused the interest of research workers for almost two centuries, and each generation of investigators has added to our knowledge in this field.

The first to describe sciatica as a disease was COTUNNUS (1764). He distinguished 3 types of the disease namely arthritic sciatica in which pain was limited to the hip joint, nervous posterior and anterior sciatica in which pain was confined to the posterior and anterior parts respectively of the leg. He described muscular atrophy and ascribed the symptoms to neuritis with oedema of the nerve sheath and consequent compression of the nerve. He drew attention to the neural origin of the disease which distinguished it clearly from joint pain.

A hundred years later (1864) LASÉGUE described his now well known leg raising test.

40 years later (1904) the Frenchmen LORTAT JACOB and SABATÉNIÉ showed that in certain cases of sciatica "sensory disturbances presented a segmentary distribution" and they introduced the term "sciatique radiculaire" which gained a firm footing by the work of DEJERINE (1916). Reports on the frequency of radicular sciatica in relation to sciatica in general vary. DEJERINE & REGNIER (1912) regarded the form as very common. WEPPEL & SALOMONSON (1911) believed that two thirds of all cases of sciatica were of radicular type and LARI & SCHAEFFER (1916) expressed the view that sciatica is most commonly of radicular origin. STURSBURG (1910) propounded that radicular sciatica was a correct diagnosis if sensibility

disorders of segmentary types could be demonstrated and that a probable diagnosis could be made if other symptoms were found such as muscular atrophy weakness of certain muscle groups etc

The aetiology of sciatica has been the subject of much speculation and it was not until 1934 that MIXTER & BARR showed that root compression due to disk herniation in the lumbar region was the commonest cause of sciatica

Many years previously however cases of intraspinal disk herniation had been published but they had fallen into oblivion For example KEY (1838) reported a case of intraspinal disk herniation from the 11th thoracic interspace the disease resulted in paraplegia and death LUSCHKA (1858) presented 2 cases of intraspinal disk herniation and demonstrated their relation to nucleus pulposus This herniation produced no symptoms but LUSCHKA claimed that if these formations had grown they could have caused pressure of the spinal cord

But as mentioned LUSCHKA's observation passed unnoticed or into oblivion because with advances in intraspinal surgery these symptomatic disk herniations became accessible to operation and they were then regarded as tumours of the intervertebral disk They were usually classified as chondroma or fibrochondroma (OPPENHEIM & KRAUSE (1909) ADSON & OTT (1922) EISEBERG (1925) ALAJOUANINE & PETIT DUTAILLIS (1929) BUCY (1930))

The first to report a case of traumatic dislocation of an intervertebral disk was KOCHER (1896) A 26 year old man had fallen from a height of 100 feet in a standing position At autopsy the disk between LI and LII showed a large bulge but no vertebral fracture was demonstrable KOCHER believed the change to be due not to protrusion but to proliferation

In 1911 MIDDLETON & TEACHER described a case A 38 year old man felt something snap in the back when lifting a heavy weight Local pain and paraplegia of the legs developed during the following night The patient died 16 days later from urinary tract infection Post mortem examination showed retropulsion of the disk between the 12th thoracic and 1st lumbar vertebrae



in the spinal canal. The mass was white and firm and particularly resembled the central part of the intervertebral disk.

The same year (1911) GOLDBWAIT published a paper in which he tried to find a relationship between sciatia and lumbosacral anomalies sacralization lumbalization spinal bifida and with the development of irregularities on the small joint surfaces of the arches. He soon discovered however that many patients with congenital anomalies had not sciatia. GOLDBWAIT concluded that subluxation of lumbosacral joints was the commonest cause of sciatia.

In 1916 ELSBACH described extradural chondromata in the spinal canal and in 1925 he reported 3 cases of extradural chondromas that he had operated upon. The patients had been treated for several years for sciatia. Atrophy of the bone musculature paresis of the peroneus and weakness of the dorsal flexion of the foot were noted.

In 1927 SCHMORL described "Knorpelknötchen" ruptures of the nucleus pulposus into the spongia. 2 years later SCHMORL & ANDRAE described also hintere Knorpelknötchen ruptures of the nucleus pulposus into the spinal canal. They did not, however believe these changes to be of clinical importance. In 1932 SCHMORL & JUNGHAUS assumed that ruptures might cause compression of the spinal cord if the ruptures assumed considerable dimensions.

Some authors felt that an affection of the musculature might be the primary disorder responsible for sciatia e.g. PITRIN (1908). This opinion had been propounded earlier by HILL DAY (1876) in his paper "On myitis chronica". PITRIN suggested that particularly in piriformis because of its close relationship to the nerve trunk might be capable of affecting the nerve. Several other authors have described this muscle as a possible cause of sciatia (FRIBERG & VINKE (1934) FRIBERG (1937) BRAYTON & ANSON (1938) HAGGART (1938)).

In 1920 HELWEG postulated that the pathological anatomical cause of the disease was myopathy probably of ischemic origin due to muscular hyperfunction. INGVAR (1936) shared his view. LINDSTEDT (1920 1922 1937) inclined to the view

that sciatica was neuralgia due to chronic irritation *e.g.* muscular hyperfunction ÅKERBLOM (1908) more or less shared LINDSTEDT's opinion

The theoretical speculations on the aetiology of sciatica also included the doctrine of the viscerosensory reflex (MACKENZIE 1893) According to that theory sciatic pain is a sort of referred pain (KELLGREN (1938) LEWIS (1942)) from pathological changes in various joints in the lumbosacral region or from the surroundings of the joints (LJUNGBAHL (1919) SMITH PETERSEN & ROGER (1926) BURT (1934) WAGNER (1935) PITKIN & PHEASANT (1936) BROCHER (1936) KIMBERLEY (1937) BADCLEY (1937 1941) STÄNDLER (1940) SCHÖBER (1940))

CRAIG & WALSH (1941) claimed While it is probably true that reflex pain exists in the leg still with our present knowledge it is difficult to estimate its frequency and an intraspinal affection certainly must be excluded before the deduction can be made that the patient's pain is reflectory Even though a number of sciatic symptoms can be explained by a referred mechanism such as muscle tenderness it is of course difficult to explain the neural loss symptoms Some of the above mentioned authors (BURT BADGLEY STÄNDLER) are also of the opinion that in those cases where neural loss symptoms are demonstrable this loss might suggest injury of a nerve root

Anomalies and pathological changes in the sacro iliac joints and in the lumbosacral region have been held responsible for sciatica particularly by orthopaedic surgeons FRIBERG (1941) reviewed papers by various authors in this field Arthritic changes in the sacro iliac joints were regarded by many authors as being capable of compressing the lumbosacral cord (GOLDTHWAIT & OSGOOD (1905) ROGERS (1911) YEOMAN (1928) KLEINBERG (1929) SASHIN (1930)) but changes in the lumbosacral region were also held responsible for sciatic pain by the pressure they exerted on different nerve roots (BAUMAN (1924) DANFORTH & WILSON (1925) KLEINBERG (1929) AYERS (1929) HIBBS & SWIFT (1929) WILLIAMS (1932) GHORMLEY (1933) SMITH (1934) KIMBERLEY (1937) MAYERDING (1938))

As known sciatica has often been ascribed to a radicular affection (LORTAT JACOB (1904)) SICARD (1918) felt that the site of affection of the roots is situated peripheral to the spinal ganglion and is due to changes in the intervertebral foramen which by pressure against the spinal nerve gives rise to sciatica. He called this form of sciatica "sciatique funiculaire", and ROGER (1930) felt that this form of sciatica is the commonest and that changes in the intervertebral foramen are of rheumatic origin.

FOERSTER (1936) also expressed the view that some cases of sciatica may be due to arthritic changes in the intervertebral foramen between the 5th lumbar vertebrae and the sacrum. This view has been shared by HEUMAN (1920) in Sweden. PUTTI (1927) shared SICARD'S view that sciatica is a form of vertebral arthritis. SICARD (1918) claimed that the common form of sciatica is funiculitis in which the IIIrd and IVth and Vth lumbar and 1st sacral roots were affected while lumbago is a funiculitis in which the IIrd, IIIrd and IVth lumbar roots were involved. This relation between sciatica and lumbago has been discussed earlier by ROMBERG (1853) and GOWERS (1892) and particularly by GARA (1907) who also stressed that tenderness to pressure over the spinal process of the 1st lumbar vertebra is a common symptom of sciatica (even in such cases in which sciatica is not preceded by lumbago). He regarded this symptom as a support for the opinion that sciatica is a root affection. In Sweden LINDSTEDT (1922) clearly showed the relationship between sciatica and lumbago.

In 1925 DANFORTH & WILSON claimed on the basis of a clinical and anatomical investigation that sciatic pain is a symptom of injury to the lower lumbar region commonly localized to the junction between LV and sacrum and that the LV root is the one primarily involved.

In 1931 STENSTRÖM performed a clinical analysis of so called neuritic symptoms in sciatica and arrived at the conclusion that in a fair number of cases of sciatica there was nerve injury apparently localized to the spinal nerves at the very site where they enter the sacral plexus. He felt that it

was not certain whether this form of sciatica could be distinguished pathogenetically from other forms of the disease

EKVALL (1939) discovered the neuritic symptom in 90 % of his cases and stressed the importance of careful neurological examination. He believed that the site of attack was radicular.

After MIXTER & BARRS (1934) renowned work had been published many other series of operated disk herniations were reported. BARR, HAMPTON & MIXTER published (1937) 58 cases and LOVE & WALSH 100 cases the following year. In 1940 LOVE & WALSH reported on 500 cases and stated that 96 % of the disk herniations were situated in the lower lumbar region. In 1947 LOVE extended his operated material to 1217 cases. LOVE published other series together with CAMP (1937) and WALSH published together with CRAIG (1941). Among other American investigators who have collected operated cases mention might be made of DANDY, NAFZIGER, INMAN & SAUNDERS, BRADFORD & SPURLING, SPURLING & BRADFORD, SPURLING & GRANTHAM.

Large series of operated cases were also collected in Europe (BUSCH & CHRISTENSEN, PENNYBACKER, DE SÈZE, O'CONNEL, FRIBERG, SJÖQVIST, MALMROS, WIBERG, NORLEN, WARIS, RÖVIG, KNUTSSON & WIBERG, HANRAETZ).

Of these NAFZIGER et al (1938) showed that ligamentum flavum might also be the cause of root compression, a view shared by other neurosurgeons (BARR, MALMROS). Other authors who have found a thickening of the ligamentum flavum to be a cause of compression are ELSBERG, FLORES, TOWNE & REICHERT, PUUSEPP, ABBOT, SPURLING, MAYFIELD & ROGERS, BROWN, BRADFORD & SPURLING and LOVE. In 1940 PENNYBACKER stressed that certain myelographic findings may be explained by hypertrophy of the ligament, but he never found any such case in which the sciatic syndrome could be ascribed entirely to hypertrophy of the ligament. It was soon realized that in some cases of sciatica operated upon no disk herniation could be found at operation. In Sweden it was in particular FRIBERG (1941) and LINDBLOM (1941) who by anatomical examinations demonstrated that root compression might

This was first recorded by DENNY BROWN (1929) and the same year by ADRIAN & BROWN independently of one another. Later further observations were made of the muscle action potentials by ECCLES & SHERRINGTON (1930) CLARK (1931) LINDSLEY (1935) and HOEFER & PUTNAM (1939).

The use of electromyography in the diagnosis of pathological changes in human beings was described in 1938 by DENNY BROWN & PENNYBACKER. The clinical use of the method is based to a large extent on the discovery of the denervation fibrillations. The first to observe fibrillations in denervated muscle was SCHIFF (1851). After having excised the n. hypoglossus on dogs he observed 5 days later fibrillations in the affected muscle. He believed the fibrillations observed in the denervated muscle to be due to muscular activity and to degeneration of the nerve.

Denervation fibrillations were rediscovered by LANGLEY & KATO (1915).

The first clinical observations were made by PROBSTER (1928) who described denervation fibrillations which he had recorded from an affected muscle in a 13 year old boy with injury to the plexus brachialis. He observed that these fibrillations did not occur in normal muscle on the opposite side. He also described denervation potentials from patients with poliomyelitis.

Later investigations of denervation fibrillations have been performed by BROWN (1937) DENNY BROWN & PENNYBACKER (1938) HOEFER & PUTNAM (1939).

WEDDELL, FEINSTEIN & PATTLE (1944) observed that fibrillations occurred in human beings 18--21 days after the denervation.

These studies led to investigations with electromyography of the lower motor neuron disease. GOLSTEN & IZZELL (1947) JASPER & JOHNSTON (1945) JASPER (1945) and LOOFBOLOPPOW (1948) demonstrated that electromyography can record the electric abnormalities accompanying lower neuron disease. The clinical use of the method as the diagnostic tool in the investigation of neurological diseases has been stressed by

HOEFER & GUTTMAN (1944) BRAZZIER WATKINS & MICHELSEN (1946) KUGELBERG (1947) and HUDDLESTON & GOLSETH (1948)

It is BUCHTHAL who must be credited most with the development of electromyography and the range of application of the method. Together with his co workers BUCHTHAL has published numerous papers in this field.

Concerning electromyography as a diagnostic adjunct in lumbar nerve root compression the reader is referred to chapter III subheading Electromyographic findings. Of authors who have published works in this field mention might be made of SHEA WOODS & WERDEN (1950) GOLSETH (1950) VON HAGEN (1955) MARINACCI (1954 1955 1958) SHEA & WOODS (1955 1956) CRUE PUDENZ & SHELDEN (1957) BONNER & SCHMIDT (1957) MENDELSON & SOLA (1958) and KNUTSSON (1958).

## Chapter III

# MATERIAL

### Classification

The material consisted of 205 patients (124 males [60.49 %] and 81 females [39.51 %]) operated upon for herniated intervertebral disks at the Orthopedic Department University Hospital of Lund during the years 1958 and 1959. One of the patients was operated upon twice.

(During the above mentioned 2 years a total of 227 operations were performed for intervertebral disk herniation (115 in 1958 and 112 in 1959). In 13 of these cases electromyography was not done owing to the absence of the author at the time of admission of these patients to the Department. In a further 8 cases myelography was not performed so that the number of patients submitted to clinical examination and myelography as well as electromyography was reduced to 205 including the patient operated upon twice thus bringing the number of operations up to 206.)

The material was divided into 2 groups.

*Group 1* — This group consisted of 182 patients who had not been operated upon previously.

*Group 2* — This group consisted of 23 patients who had been operated upon previously. One of the patients was operated upon twice.

The operative findings made in the 182 patients in group 1 were as follows:

| Findings   | Total | Males | Females |
|--|-------|-------|---------|
| a) disk herniation between L <sub>1</sub> and S <sub>1</sub> | 60    | 36    | 24      |
| b) disk herniation between L <sub>1</sub> and L <sub>2</sub> | 1     | 1     | —       |
| c) disk herniation between L <sub>2</sub> and L <sub>3</sub> | 74    | 40    | 28      |
| d) disk herniation between L <sub>3</sub> and L <sub>4</sub> | 2     | 1     | 1       |
| e) protrusion between L <sub>1</sub> and S <sub>1</sub>      | 11    | 6     | 5       |
| f) protrusion between L <sub>2</sub> and L <sub>3</sub>      | 14    | 7     | 7       |
| g) negative  | 20    | 13    | 7       |

Group 2 consisted of 23 patients operated upon a total of 24 times for disk herniation and the operative findings are given below

| Findings   | Total | Males | Females |
|--|-------|-------|---------|
| a) herniation between L <sub>4</sub> and S <sub>1</sub>                                      | 6     | 5     | 1       |
| b) herniation between L <sub>4</sub> and L <sub>5</sub>                                      | 4     | 3     | 1       |
| c) herniation between L <sub>3</sub> and L <sub>4</sub>                                      | 1     | —     | 1       |
| d) protrusion between L <sub>4</sub> and S <sub>1</sub>                                      | 3     | 1     | 2       |
| e) protrusion between L <sub>4</sub> and L <sub>5</sub>                                      | 1     | —     | 1       |
| f) protrusion between L <sub>3</sub> and L <sub>4</sub>                                      | 1     | 1     | —       |
| g) adhesions between L <sub>4</sub> and S <sub>1</sub>                                       | 3     | —     | 3       |
| h) adhesions between L <sub>4</sub> and L <sub>5</sub>                                       | 2     | 2     | —       |
| i) adhesions between L <sub>4</sub> and S <sub>1</sub> and L <sub>4</sub> and L <sub>5</sub> | 2     | 2     | —       |
| j) negative  | 1     | 1     | —       |

Of the 20 patients in whom the operative findings were negative (group 1 subheading g) there were 7 with osteophytes which thus provided a satisfactory explanation for the root symptoms in 1 the space was narrow and 4 had pronounced varices. In the remaining 8 cases exploration revealed no signs at all of a pathological condition.

A comparison of these operative findings with those reported by earlier investigators is given below

#### *Intervertebral disk herniation*

|                               |      |   |           |
|-------------------------------|------|---|-----------|
| 1941 FRIBERG                  | 6    | % | (11/28)   |
| 1942 MALMROS                  | 83   | % | (100/118) |
| 1944 SHINNERS & HAMBY         | 83   | % | (116/140) |
| 1946 SEYING & SJÖQVIST        | 74   | % | (309/403) |
| 1948 WARIS                    | ~    | % | (289/374) |
| 1961 Author's group 1         | 12.3 | % | (137/182) |
| 1961 Author's entire material | 71.8 | % | (148/206) |

#### *All disk lesions (protrusions included)*

|                               |      |   |           |
|-------------------------------|------|---|-----------|
| 1941 FRIBERG                  | 88   | % | (21/28)   |
| 1942 MALMROS                  | 83   | % | (100/118) |
| 1944 SHINNERS & HAMBY         | 83   | % | (116/140) |
| 1946 SEYING & SJÖQVIST        | 83   | % | (342/403) |
| 1948 WARIS                    | 88   | % | (330/374) |
| 1961 Author's group 1         | 89   | % | (167/182) |
| 1961 Author's entire material | 86.2 | % | (187/206) |



*Other probable causes of root compression*

|                               |       |          |
|-------------------------------|-------|----------|
| 1911 FRIBERG                  | 7 %   | (4/58)   |
| 1912 MALMIOS                  | 13 %  | (15/118) |
| 1916 SENNING & SJÖQVIST       | 2 %   | (8/403)  |
| 1918 WARIS                    | 4 %   | (16/374) |
| 1961 Author's group 1         | 6.6 % | (12/182) |
| 1961 Author's entire material | 9.9 % | (19/206) |

*Negative findings*

|                               |       |          |
|-------------------------------|-------|----------|
| 1911 FRIBERG                  | 5 %   | (3/58)   |
| 1912 MALMIOS                  | 3 %   | (3/118)  |
| 1944 SHINNERS & HAMBY         | 17 %  | (24/140) |
| 1916 SENNING & SJÖQVIST       | 13 %  | (53/403) |
| 1918 WARIS                    | 4 %   | (16/374) |
| 1961 Author's group 1         | 4.4 % | (8/182)  |
| 1961 Author's entire material | 4.4 % | (9/206)  |

There is general agreement that the 2 lowest lumbar disks are those most commonly herniated. This was also found in the present material.

The cases are listed according to the side involved below.

*Group 1*

| Findings                                    | Males | Females   |
|---|-------|-----------|
| a) 34 left sided                            | 29    | 12        |
| 26 right sided                              | 14    | 12        |
| b) 1 right sided                            | 1     | —         |
| c) 46 left sided                            | 29    | 17        |
| 28 right sided                              | 17    | 11        |
| d) 1 left sided                             | 1     | —         |
| 1 right sided                               | —     | 1         |
| e) 9 left sided                             | 6     | 3         |
| 2 right sided                               | 1     | 1         |
| f) 9 left sided                             | 3     | 6         |
| 5 right sided                               | 4     | 1         |
| g) 11 left sided                            | 7     | 4         |
| 9 right sided                               | 6     | 3         |
| Total number of left sided disk herniations | 81    | 52 m 29 f |
| " " right sided                             | 6     | 32 m 21 f |
| " " left sided protrusions                  | 18    | 9 m 9 f   |
| " " right sided                             | 7     | 5 m 2 f   |

## Group 9

|    | Flu lungs     | Males | Females |
|----|---------------|-------|---------|
| a) | 3 left sided  | 2     | 1       |
|    | 3 right sided | 3     | —       |
| b) | 4 left sided  | 3     | 1       |
| c) | 1 left sided  | —     | 1       |
| d) | 2 left sided  | 1     | 1       |
|    | 1 right sided | —     | 1       |
| e) | 1 left sided  | —     | 1       |
| f) | 1 left sided  | 1     | —       |
| g) | 2 left sided  | —     | 2       |
|    | 1 right sided | —     | 1       |
| h) | 2 left sided  | 2     | —       |
| i) | 2 right sided | 2     | —       |
| j) | 1 right sided | 1     | —       |

Total number of left sided disk herniations 8 5 m 3 f  
 right sided 3 3 m  
 left sided protrusions 4 2 m 2 f  
 right sided 1 1 m  
 left sided adhesions 4 2 m 2 f  
 right sided 3 2 m 1 f

Of the 206 cases the symptoms were left sided in 111 (53.9 %) and right sided in 70 (36.4 %) while in 20 (9.7 %) they were bilateral. Of these 20 the symptoms were predominant on the left side in 10 and on the right side in 10. Thus the symptoms were predominant on the left side in 126 (61.2 %) and on the right side in 80 (38.8 %). As mentioned in the present material males were predominant (60.49 against 39.51 % females). This preponderance is in agreement with that found in most previous series [LOVE & WALSH (1940) 71 % MALMIROS (1942) 63 % WARIS (1949) 72 % RÖVIG (1949) 70 % BARR & MEXTER (1941) 78 % and FRIBERG (1941) 61.4 %].

In the present material the left side was involved more frequently than the right. This observation has also been made by earlier authors [MALMIROS (1942) 62 % YASKIN et al (1944)

## The age distribution

|             | Diskernition |         |         |         |         |         | I retrusion<br>I A S I | I retrusion<br>I A I V | Negative | Total<br>Group 1 | Group 2 | I nitre<br>material |
|-------------|--------------|---------|---------|---------|---------|---------|------------------------|------------------------|----------|------------------|---------|---------------------|
|             | I A S I      | I A I V | I A I V | I A I V | I A I V | I A I V |                        |                        |          |                  |         |                     |
| 10-19 years | 1            | —       | —       | 1       | 1       | —       | —                      | —                      | —        | 2                | —       | 1                   |
| 20-29       | 7            | 4       | —       | 4       | 2       | —       | 1                      | 1                      | —        | 13               | 1       | 14                  |
| 30-39 "     | 12           | 7       | 1       | —       | 7       | —       | 1                      | 3                      | 2        | 31               | 7       | 38                  |
| 40-49       | 9            | 9       | —       | —       | 10      | —       | 3                      | 1                      | 2        | 33               | 3       | 36                  |
| 50-59       | 6            | 4       | —       | —       | 4       | 1       | —                      | 3                      | 2        | 26               | 4       | 30                  |
| 60-69       | 1            | —       | —       | —       | 3       | —       | 1                      | —                      | 1        | 4                | —       | 5                   |
| 70-79       | —            | —       | —       | —       | 1       | —       | —                      | —                      | —        | —                | —       | —                   |
| Total       | 36           | 24      | 1       | 40      | 28      | 1       | 6                      | 5                      | 13       | 110              | 15      | 125                 |
|             | M            | I       | M       | I       | F       | M       | M                      | F                      | M        | I                | M       | I                   |

## A comparison with other materials

| Age         | I over 1947<br>of 1217 cases | Watts 1948<br>of 374 cases | I nitro 1-<br>Will erg 1948<br>of 201 cases | I nitro 1-<br>of 66 cases |
|-------------|------------------------------|----------------------------|---|---------------------------|
| 10-19 years | 21                           | —                          | 32  | 14                        |
| 20-29       | 103                          | 18                         | 115   | 121                       |
| 30-39       | 353                          | 43                         | 307   | 286                       |
| 40-49       | 305                          | 28                         | 343   | 325                       |
| 50-59       | 131                          | 10                         | 151   | 204                       |
| 60-69       | 27                           | 1                          | 52  | 44                        |
| 70-79       | —                            | —                          | —   | 05                        |

TABLE 2  
*Duration of the symptoms*

|                         | Less<br>than<br>3 weeks | 1-3<br>months | 4-6<br>months | 7-1<br>months | More<br>than<br>1 year |
|-------------------------|-------------------------|---------------|---------------|---------------|------------------------|
| Disk herniation L V-S I | —                       | 20            | 21            | 13            | 6                      |
| L V-L VI                | —                       | 1             | —             | —             | —                      |
| L IV-L V                | 1                       | 28            | 26            | 10            | 9                      |
| L III-L IV              | —                       | 2             | —             | —             | —                      |
| Protrusion L V-S I      | —                       | 3             | 1             | 4             | 3                      |
| Protrusion L IV-L V     | —                       | 2             | 4             | 6             | 2                      |
| Negative findings       | —                       | 5             | 6             | 3             | 6                      |
| Group 2                 | 1                       | 5             | 9             | 6             | 3                      |
| Total                   | 2                       | 66            | 67            | 42            | 29                     |

58 % PETITS DUTAILLIS & DE SEZE (1945) 59 % and WARIS (1949) 51 % RÖVIG (1949) however found the disease to be slightly more common on the right side (50 right sided 45 left sided and 4 bilateral)]

As to the ratio between unilateral and bilateral projection of pain it was unilateral in 90.3 % of the present material and bilateral in 9.7 %. A similar ratio was given by BRADFORD & SPURLING (1945) namely 8 % bilateral sciatica while most other authors give a somewhat higher figure for bilateral radiation of pain *e.g.* LOVE & WALSH (1938) 16 % BARR & MINTZ (1941) 20 % FRIBERG (1941) 22.7 % and MACEY (1940) 12 %

The age distribution of the material is given in Table 1 and the duration of the symptoms in Table 2

### Projection of pain

Sciatic pain and its projection has been the subject of discussion by many authors

In 1841 VALLEIX distinguished 2 types of sciatic pain namely spontaneous and provoked. According to him provoked pain consisted of pain produced by compression of the nerve for example cough and special bodily movements. Pain produced

the thigh and 1 patient reported pain even on the posterior surface of the leg and down into the great toe

Since the results of NORLÉN's clinical investigations did not agree entirely with LÖRSTER's assertion NORLÉN performed an experimental investigation. He irritated the 4th and 5th lumbar roots as well as the 1st and 2nd sacral roots mechanically at operation and reported the following findings

1 On irritation of the 4th lumbar root (14 cases) the patients reported that they felt projected pain in the hip and the anterior surface and inner side of the thigh down to the knee and several also reported pain on the anterior side of the lower leg. 2 also described pain in the calf. None of them reported projection of pain into the foot or great toe

2 On irritation of the 5th lumbar root (16 cases) 14 patients reported that they felt projected pain on the posterior side of the leg and in the great toe. 2 of the patients described pain radiating out into the foot without saying where in the foot

3 In 13 cases the 1st sacral root was irritated. Of these patients 20 reported that they felt radiating pain on the back of the leg down to the heel. 3 described pain in the foot without saying where. 3 claimed pain in the entire foot and lateral border of the foot and 1 pain down to the ankle but not into the foot. In 1 case a patient reported pain down to the back of the knee but not further

4 The 2nd sacral root was irritated in 10 cases. 7 of them reported projection of pain to the back of the thigh down to the back of the knee. 2 described pain on the back of the leg to just below the knee and 1 down to the foot but did not say where

In WARIS material from 1948 42 % (64 of 153) of the herniations between IV and SI described projection of pain resembling NORLÉN's SI syndrome. In only 1 of the cases of herniation at this level was pain projected out into the great toe. In 3 into the perineum and in 56 % (85 of 153) the pain was less defined and radiated into the thigh calf or ankles

Of patients with herniation between LIV and IV 21 % (27 of 129) reported projection of pain into the great toe (typical

of compression of the 5th lumbar root) 17 % (22 of 129) reported pain in the heel (characteristic of compression of the 1st sacral root) In 62 % of the cases (80 of 129) pain was diffuse and radiated mainly down to the calf

TABLE 3  
*Projection of pain*

|                        | Hip | Thigh | Knee | Calf | Ankles | Foot | 1st toes | Great toe       | Heel            |     |
|------------------------|-----|-------|------|------|--------|------|----------|-----------------|-----------------|-----|
| <b>Group 1</b>         |     |       |      |      |        |      |          |                 |                 |     |
| Herniated disk between |     |       |      |      |        |      |          |                 |                 |     |
| L V—S I                | 3   | —     | 4    | 15   | 3      | 10   | 10       | 3 <sup>1</sup>  | 19 <sup>2</sup> | 60  |
| L V—I V I              | —   | —     | —    | —    | —      | —    | —        | —               | 1               | 1   |
| L IV—L V               | —   | 3     | 4    | 17   | 11     | 10   | 7        | 16 <sup>3</sup> | 6               | 74  |
| L III—L IV             | —   | —     | 2    | —    | —      | —    | —        | —               | —               | 9   |
| Protrusion L V—S I     | —   | —     | —    | 3    | 2      | 2    | 1        | —               | 3               | 11  |
| I IV—L V               | —   | —     | 1    | 6    | 1      | 5    | 1        | —               | —               | 14  |
| Negative               | 1   | —     | 2    | 5    | 1      | 5    | 9        | 3               | 1               | 20  |
| Total group 1          | 4   | 3     | 13   | 46   | 18     | 32   | 21       | 22              | 23              | 182 |
| <b>Group 2</b>         |     |       |      |      |        |      |          |                 |                 |     |
| Herniated disk         |     |       |      |      |        |      |          |                 |                 |     |
| L V—S I                | —   | —     | —    | 1    | —      | —    | —        | 2               | 3               | 6   |
| L IV—L V               | —   | —     | —    | —    | 2      | 1    | —        | —               | 1               | 4   |
| L III—L IV             | —   | —     | —    | —    | —      | —    | —        | 1               | —               | 1   |
| Protrusion L V—S I     | 1   | —     | —    | —    | 1      | —    | —        | —               | 1               | 3   |
| L IV—L V               | —   | —     | —    | —    | —      | 1    | —        | —               | —               | 1   |
| L III—L IV             | —   | —     | —    | —    | —      | —    | 1        | —               | —               | 1   |
| Adhesions L V—S I      | —   | —     | —    | 2    | —      | —    | 1        | —               | —               | 3   |
| L IV—L V               | —   | —     | —    | —    | —      | 1    | —        | 1               | —               | 2   |
| L V—S I +              | —   | —     | —    | —    | —      | —    | —        | —               | —               | —   |
| + L IV—L V             | —   | —     | —    | 1    | —      | —    | —        | 1               | —               | 2   |
| Negative expl          | —   | —     | —    | —    | —      | —    | —        | —               | 1               | 1   |
| Total group 2          | 1   | —     | —    | 1    | 3      | 3    | 2        | 5               | 6               | 24  |
| Entire material total  | 5   | 3     | 13   | 50   | 21     | 35   | 23       | 27              | 29              | 206 |

3 % projected pain into the great toe

20 % projected pain into the heel

21.6 % projected pain into the great toe

8.1 % projected pain into the heel

the thigh and 1 patient reported pain even on the posterior surface of the leg and down into the great toe

Since the results of NORLEN's clinical investigations did not agree entirely with FOERSTER's assertion NORLEN performed an experimental investigation. He irritated the 4th and 5th lumbar as well as the 1st and 2nd sacral roots mechanically. The following findings

(14 cases) the patients and the an

Page 63 line 6 LV cases should read LV+SV  
 Page 63 line 29 14 should read LV+SV cases  
 Page 68 fig 3 Black fields indicating the occurrence of denervation potentials in ext dig. brev. LV 3 cases  
 Page 69 fig 4 Black fields indicating the occurrence of denervation potentials in case number 1638 28 are missing for cases 40, 58 muscles from m. tensor fasciae latae to m. erector trunci  
 Page 106 in the table below Others SV+SV should read SV+SV  
 Page 113 line 12 myelography showed abnormalities should read myelography showed no abnormalities  
 Page 121 line 7 186% should read 78.3%

se  
 ck  
 th  
 eral  
 into  
 back

f them  
 down to  
 ne leg to  
 not say

3) of the  
 on of pun  
 he cases of  
 do the great  
 53) the pun  
 f or ankles  
 LV 21 % (27  
 at toe (typical

16 also had paresis of the great toe and in 5 the patellar reflex was weak or absent. In the other 3 cases the weakness or absence of the Achilles tendon reflex was also accompanied by weakness or absence of the patellar reflex and paresis of the great toe.

b) *Disk herniation between LV and LVI* —

c) *Disk herniation between LIV and LV* — Of the 74 cases of herniation of the disk between LIV and LV the Achilles tendon reflex was weak or absent in 28. It was the only finding in 9; it was combined with weakness or absence of the patellar reflex as well as paresis of the great toe in 3; but only with paresis of the great toe in 16 and only with weakness or absence of the patellar reflex in 1.

d) *Disk herniation between LIII and LIV* —

e) *Protrusion between LV and SI* — Of the 11 cases of protrusion between LV and SI the Achilles tendon reflex was weak or absent in 8. In 1 of these cases it was combined with weakness or absence of the patellar reflex and great toe paresis and in 2 with paresis of the great toe while in 2 it was the only finding.

f) *Protrusion between LIV and LV* — Weakness or absence of the Achilles tendon reflex was the only finding in 1 case of the 14 cases of protrusion between LIV and LV and it was combined with paresis of the great toe in 2 cases.

g) *Negative findings at operation* — Of the 20 cases in which the findings at operation showed no disk pathology weakness or absence of the Achilles tendon reflex was the only finding in 4; it was combined with paresis of the great toe in 3 and with weakness of the patellar reflex as well as paresis of the great toe in 1 case.

## B Group 2

a) *Disk herniation between LV and SI* — Of the 6 cases of disk herniation between LV and SI the Achilles tendon reflex was weak or absent in 5. This weakness or absence of the Achilles tendon reflex was the only finding in 2 while it was combined with weakness of the great toe in 2 and with weak



ness of the patellar reflex as well as weakness of the great toe in 1

b) *Disk herniation between LIV and LV* — Of the 4 cases of disk herniation between LIV and LV the Achilles tendon reflex was weak or absent in 3. It was the only finding in 1 case while in 1 it was combined with weakness of the great toe and in 1 with weakness of the patellar reflex.

c) *Disk herniation between LIII and LIV* — Herniation at this level was seen in 1 case and in that one the weakness of the Achilles tendon reflex was accompanied by paresis of the great toe and weakness of the patellar reflex.

d) *Protrusion between LV and SI* — Of the 3 patients with protrusions between LV and SI the Achilles tendon reflex was weak or absent in all. It was the only change in 2 while it was combined with weakness of the great toe in 1.

e) *Protrusion between LIV and LV* —

f) *Protrusion between LIII and LIV* — This was seen in 1 case. The Achilles tendon reflex was weak and was associated with ready fatigability of the great toe and weakness of the patellar reflex.

g) *Adhesions between IV and SI* — This was seen in 3 cases. In 2 of them the Achilles tendon reflex was weak or absent and in both it was combined with weakness of the great toe.

h) *Adhesions between LIV and LV* — This was seen in 2 cases. In 1 of them the Achilles tendon reflex was absent and the great toe was weak.

i) *Adhesions between LIV and IV + IV and SI* — Adhesions were seen in 2 cases. In 1 of them there was weakness of the Achilles tendon reflex and of the great toe.

j) *Negative finding at operation* — This subgroup consisted of a single case. There was possibly slight weakness of the Achilles tendon reflex.

The literature on changes in the Achilles tendon reflex in patients with sciatica is abundant.

STERNBLER (1893) was probably the first to report a case of sciatica in which the Achilles tendon reflex was absent.

BABINSKI (1896) expressed the view that the absence of the Achilles tendon reflex in sciatica was evidence of inflammation of the sciatic nerve in such cases

BABINSKI like BIRO (1897) reported areflexia in patients without measurable atrophy or weakness of the calf muscles. Likewise the Achilles tendon reflex could be absent in patients with normal sensibility

ACCORNEO (1914) also found that some cases which appeared to be severe with muscular atrophy and sensibility disorders showed normal Achilles tendon reflex while others which appeared to be slight cases showed areflexia

How often is the Achilles tendon reflex absent in sciatica?

The frequency with which the Achilles tendon reflex has been found to be absent in sciatica due to intervertebral disk herniation varies somewhat from one author to another. As a rule the authors did not state whether the reflex was weak or absent and data on the site or level of the herniation in patients with areflexia are incomplete and scanty

For example LOVE & WALSH (1938) stated that the reflex was weak or absent in 57 % of their cases and in 1940 the same authors gave 60 %. BRADFORD & SPURLING (1939) found the reflex to be weak or absent in 15 of 30 cases. In 10 of these cases the herniation was located between L<sub>4</sub> and S<sub>1</sub> and in 5 between L<sub>4</sub> and L<sub>5</sub>. BARR (1938) found the Achilles tendon reflex to be absent in 50 % of his cases and in 1941 BARR & MIXTER reported weakness or absence of the Achilles tendon reflex in 70 % of their cases. PENNYBACKER found the Achilles tendon reflex to be normal in only 5 of his 28 cases but on analysis of his series it will be seen that he had 23 cases of herniation of the disk between L<sub>4</sub> and S<sub>1</sub> and only 5 between L<sub>4</sub> and L<sub>5</sub>. In 1942 SJÖQVIST found the Achilles tendon reflex to be absent in 12 and weak in 5 of a total of 17 cases of herniation of the disk between L<sub>4</sub> and S<sub>1</sub>. In 4 cases of disk herniation between L<sub>4</sub> and L<sub>5</sub> 2 had a normal Achilles tendon reflex and in 2 it was weak. In MALMROS series of 90 cases areflexia was noted in 38 (bilateral in 11) and weakness in 18

WIBERG (1943) found the Achilles tendon reflex to be un

TABLE 6  
Changes in the Achilles tendon reflex  
(According to different authors)

|                                      | At sent | Weak | Total | Number of cases |
|--------------------------------------|---------|------|-------|-----------------|
| Disk herniation between I V and S I  |         |      |       |                 |
| 1941 CRAIG & WALSH                   | —       | —    | 68    | *               |
| 1942 MALMROS                         | —       | —    | 70    | 37              |
| 1943 WIBERG                          | 60      | 23   | 83    | 69              |
| 1944 NORLÉN                          | 57      | 32   | 89    | 46              |
| 1948 STÅHL                           | 31      | 25   | 56    | 167             |
| 1948 WARIS                           | 70      | 24   | 94    | 153             |
| 1951 FRIBERG & HULT                  | —       | —    | 80    | 124             |
| 1958 KNUTSSON & WIBERG               | —       | —    | 83    | 133             |
| 1961 Author's group 1                | 38.3    | 41.7 | 80    | 60              |
| 1961 Author's entire material        | 39.4    | 40.9 | 80.3  | 66              |
| Disk herniation between L IV and L V |         |      |       |                 |
| 1941 CRAIG & WALSH                   | —       | —    | 37    | *               |
| 1942 MALMROS                         | —       | —    | 58    | 50              |
| 1943 WIBERG                          | 32      | 22   | 54    | 68              |
| 1944 NORLÉN                          | 13      | 28   | 41    | 53              |
| 1948 STÅHL                           | 6       | 10   | 16    | 117             |
| 1948 WARIS                           | 25      | 25   | 50    | 129             |
| 1961 Author's group 1                | 10.8    | 27   | 37.8  | 74              |
| 1961 Author's entire material        | 12.8    | 26.9 | 39.7  | 78              |

\* In a series of cases in which the number of herniations in the different interspaces not is given

## II Weakness or absence of patellar reflex

### A Group 1

a) Disk herniation between L V and S I — The patellar reflex was changed in association with a change in the Achilles tendon reflex in 5 cases and together with weakness of the great toe in 1 and in combination with both of these changes in 3 patients with herniation in the lumbosacral space

b) *Disk herniation between IV and LV* — This was seen in 1 case and here a weakness of the patellar reflex was accompanied by a weakness of the great toe

c) *Disk herniation between LIV and LV* — The patellar reflex was weak or absent in combination with weakness or absence of the Achilles tendon reflex in 1 case in combination with paresis of the great toe in 5 cases and in combination with both in 3 cases

d) *Disk herniation between LIII and LIV* — This was noted in 2 cases in which a change in the patellar reflex was the only finding

e) *Protrusion between LV and SI* — The patellar reflex was changed in 1 case and then in combination with change of the Achilles tendon reflex and weakness of the great toe

f) *Protrusion between LIV and LV* — The patellar reflex was changed in combination with weakness of the great toe in 1 case

g) *Negative findings at operation* — A change in the patellar reflex was the only finding in 2 cases it was seen in association with weakness of the great toe in 4 and in combination with a changed Achilles tendon reflex and weakness of the great toe in 1

## B Group 2

a) *Disk herniation between LV and SI* — Of the 6 cases of herniation between LV and SI the patellar reflex was weak in 1 and was then combined with absence of the Achilles tendon reflex and weakness of the great toe

b) *Disk herniation between LIV and LV* — Of the 4 cases of herniation of the disk between LIV and LV the patellar reflex was absent in 1 and was then combined with absence of the Achilles tendon reflex

c) *Disk herniation between LIII and LIV* — This was seen in 1 case and in this a weakness of the patellar reflex was accompanied by a weakness of the great toe as well as a weakness of the Achilles tendon reflex

d) *Protrusion between LV and SI* —

e) *Protrusion between IIV and IV* —

f) *Protrusion between III and IIV* — This was seen in 1 case and here a weakness of the patellar reflex was accompanied by a weakness of the great toe as well as a weakness of the Achilles tendon reflex

g) *Adhesions between IV and SI* —

h) *Adhesions between IIV and IV* —

i) *Adhesions between IV and SI+IIV and IV*

j) *Negative findings* —

Only scanty data are available in the literature on the weakness or absence of the patellar reflex in sciatica or in disk herniation between IIV and IV and IV and SI. WIBERG (1943) reported weakness of the patellar reflex in 13 cases and absence in 1 of 137 cases of disk herniation of the 2 lowermost lumbar intervertebral disks

Herniations between III and IIV are very uncommon in the various series on record but it was pointed out that the patellar reflex was absent or may have been weak in these cases (SPURLING & GRANTHAM 1940, CRAIG & WALSH 1941). WIBERG (1943) reported unchanged patellar reflex in 2 cases and weakness of the reflex in 1 of a total of 3 cases of herniation between III and IIV

In NORLÉN's (1941) series of 115 cases of sciatica the patellar reflex was weak in 5. In 3 the herniation was situated between IIV and IV and 2 between IV and SI. In 2 cases of disk herniation between III and IIV the patellar reflex was weak

It has been claimed that the pathways of the patellar reflex run through the 2nd, 3rd and 4th lumbar roots (FORSTNER, OPPENHEIM, DEJERING and LUBBERG). It is therefore quite understandable why the patellar reflex is changed in the presence of disk herniation between III and IIV where it is mainly the LIV root that is exposed to compression. SHERRINGTON showed this as early as 1893 in experiments on *Macacus Rhesus*

In operations for arthrosis deformans coxae in which the sensible part of the IIV root was cut off the patellar reflex was as a rule weak and in some cases it even disappeared

completely (VON REIS SAHLGREN and SJÖQVIST 1943 NORLEN 1944)

WARIS (1948) observed distinct changes of the patellar re

TABLE 7

*Changes in the patellar reflex*  
(According to different authors)

|                               | Absent | Weak | Total | Number of cases |
|-------------------------------|--------|------|-------|-----------------|
| 1919 MALMROS                  | 1      | 8    | 9     | 90              |
| 1943 WIBERG                   | 1      | 10   | 11    | 140             |
| 1944 YASKIN & FINKELSTEIN     | —      | —    | 18    | 50              |
| 1945 BRADFORD & SPURLING      | —      | —    | > 10  | —               |
| 1948 WARIS                    | 2      | 5    | 7     | 374             |
| 1961 Author's group 1         | 22     | 143  | 165   | 182             |
| 1961 Author's entire material | 24     | 141  | 165   | 206             |

TABLE 8

*Changes in the patellar reflex*

|                               | Herniation between I III and L IV | Herniation between I IV and L V | Herniation between L V and S I |
|-------------------------------|-----------------------------------|---------------------------------|--------------------------------|
| 1941 CRAIC & WALSH            | 49                                | 90                              | 9                              |
| 1942 MALMROS                  | —                                 | 16 (800)                        | —                              |
| 1943 WIBERG                   | 33 (13)                           | 10 (14137) <sup>2</sup>         | —                              |
| 1944 NORLEN                   | 100 <sup>o</sup> (2/2)            | 3/2                             | 2/2 <sup>2</sup>               |
| 1948 WARIS absent             | 43 (3/1)                          | 3 (4129)                        | 1 (1153)                       |
| weak                          | 57 (47)                           | 3 (4129)                        | 5 (8153)                       |
| total                         | 100 (71)                          | 6 (8129)                        | 6 (9153)                       |
| 1961 Author's group 1         |                                   |                                 |                                |
| absent                        | —                                 | 27 (214)                        | —                              |
| weak                          | 100 (22)                          | 95 <sup>o</sup> (714)           | 11 (160)                       |
| total                         | 100 <sup>o</sup> (22)             | 122 (974)                       | 17 (160)                       |
| 1961 Author's entire material |                                   |                                 |                                |
| absent                        | —                                 | 38 (374)                        | —                              |
| weak                          | 100 (33)                          | 9 (778)                         | 3 (266)                        |
| total                         | 100 (33)                          | 128 (1078)                      | 3 (266)                        |

In a series of 449 cases the level of the herniations not given 4th and 5th interspace jointly

In a series of 115 cases the level of the herniations not given

flex in 27 (7 %) of his patients. The reflex was absent in 9 and weakened in 18. WARIS stated that nothing definite can be said about the inconstant changes of the patellar reflex and BRADFORD & SIURLING arrived at the same conclusion.

The results reported by previous authors and those found in the present investigation are compared below.

### III Weakness or paralysis of the great toe

#### A Group 1

a) *Disk herniation between IV and SI* — Paralysis or weakening of the great toe occurred in 28 cases of 60 cases of disk herniation between IV and SI. In 3 of these the change was accompanied by changes in the patellar and Achilles tendon reflexes: in 1 case together with a changed patellar reflex and in 15 together with a changed Achilles tendon reflex, while weakness of the great toe was the only finding in 9 cases.

b) *Disk herniation between IV and V* — 1 case and then there was weakening of the great toe in combination with slight weakness of the patellar reflex.

c) *Disk herniation between IV and V* — In the 74 cases of disk herniation between IV and V weakness of the great toe was the only finding in 33; it was combined with changes in the Achilles tendon reflex in 15, with changes in the patellar reflex in 5, and in combination with changes in the patellar reflex and the Achilles tendon reflex in 3.

d) *Disk herniation between III and IV* —

e) *Protrusion between IV and SI* — Weakness of the great toe was the only finding in 2 cases; it was seen in combination with changes of the patellar reflex as well as the Achilles tendon reflex in 1, and in combination with a change in the Achilles tendon reflex only in 2.

f) *Protrusion between IV and V* — Weakness of the great toe was the only finding in 7 cases and was seen in combination with a change in the Achilles tendon reflex in 2, and with a change in the patellar reflex in 1.

g) *Negative findings at operation* — Weakness of the great toe was the only finding in 2 cases; it was seen in combination

with a change in the patellar reflex as well as in the Achilles tendon reflex in 1 in combination with the patellar reflex only in 4 and in combination with the Achilles tendon reflex only in 3

## B Group 2

a) Of the 6 cases of *herniation between LV and SI* weakness of the great toe was noted in 4 In 1 of them this was the only change it was seen in combination with weakness of the Achilles tendon reflex in 2 and in 1 the patellar reflex was weak and the Achilles tendon reflex was absent

b) In the 4 cases of *disk herniation between LIV and LV* there was weakness of the great toe in 2 In 1 of them it was the only change and in 1 it was associated with weakness of the Achilles tendon reflex

c) *Disk herniation between LIII and LIV* — This was seen in only 1 case and here a paresis of the great toe was associated with a weakness of the patellar reflex as well as a weakness of the Achilles tendon reflex

d) *Protrusion between LV and SI* — Of the 3 protrusions between LV and SI there was weakness of the great toe in 1 and then associated with a weakness of the Achilles tendon reflex

e) *Protrusion between LIV and LV* — This was seen in 1 case and then weakness of the great toe was the only finding

f) *Protrusion between LIII and LIV* — Protrusion at this level was noted in only 1 case and here a weakness of the great toe was combined with weakness of the patellar as well as the Achilles tendon reflexes

g) *Adhesions between LV and SI* — Of these 3 cases 2 showed weakness of the great toe as well as a changed Achilles tendon reflex

h) *Adhesions between LIV and LV* — There were 2 cases and in both there was weakness of the great toe in combination with absence of the Achilles tendon reflex in 1 of them and as the only finding in 1

i) *Adhesions between LIV and LV + LV and SI* — There



were 2 cases and in both there was weakness of the great toe which was combined with weakness of the Achilles tendon reflex in 1

*g) Negative findings —*

It appears that less importance was attached to paresis in earlier until a relatively long time after MIXTER & BARR had described the causal relation between disk herniations and paresis

In 1890 GUINON & PARMENTIER reported a number of cases with loss of local motor symptoms particularly in the form of paresis and atrophy of the dorsal flexor tendons of the foot and toes

IKVALL (1939) called attention to the fact that paresis of the dorsal flexors of the great toe is by no means uncommon in sciatica

STURLING & GRANTHAM (1940) pointed out that in sciatica due to disk herniation paresis of certain groups of muscles have been observed but one had not made any detailed study of these cases

SJÖQVIST (1942) writes that in 1 of his cases he found paresis of the dorsal flexors of the great toe In that case the disk herniation was between LIV and LV

MALMROS (1942) observed that of his 90 patients 3 had nearly severe paresis of the dorsal flexors of the foot and great toe and that 1 case had isolated paresis of the dorsal flexors of the great toe In all of these patients the herniation was situated between LIV and LV MALMROS did not draw any far reaching conclusions from this observation concerning the site of the herniation

The significance of paresis of the great toe has been stressed above all by NORLÉN (1944) He found such paresis in more than 20 % of patients with herniation of the disk between LIV and LV The corresponding figure in WARIS (1948) material is 22 % (+uncertain weakness in a further 7 %) and STÅHL (1948) gave 40 % and RÖVIC (1949) 47 % IRIBERG & HULT (1951) reported 48 % and KAUTSSON & WIBERG (1958) 71 %

In the present group 1 weakness of the great toe was seen

in 56 (75.7 %) of the 74 cases of disk herniation between LIV and LV

These figures 71 % and 75.7 % respectively appear very high but in this series the strength of the dorsal flexors of the great toe was tested very carefully. The patient was thus instructed to bend the great toe not only a few times but 10—15 times and in this way it was possible to note any fatigability on the affected side which is a satisfactory sign for diagnosing weakness of the muscles.

In cases of disk herniation between LV and SI WARIS found a weakness of the great toe in 7 % STÅHL in 5.4 % and ROVIC in 12 % while in the present material it was found with such a high frequency as 46.7 % (28/60). Of these cases however weakness of the great toe in 19 was combined with reflex changes and thus weakness of the great toe was the only clinical finding in lumbosacral disk herniation in 9 (15 %) (These cases are discussed further in the chapter on Correlation with operative findings.)

Paresis and atrophy of *musculus extensor digitorum brevis* have been described by KUGELBERG & PETERSEN (1950) as a common symptom in disk herniation between LIV and LV. In a series of 41 cases of disk herniation between LIV and LV they found these symptoms in 35 (85 %) cases and weakness of the great toe in 46 % of the same cases.

As to the *musculus extensor digitorum brevis* the atrophy was either clear (23 cases) or the consistency of the muscle on maximum contraction was softer than that of the contralateral foot (12 cases).

In 35 cases electromyography also showed signs of denervation of the muscle in question. The electromyographic findings agreed with the clinical picture in all cases except 2.

A corresponding examination of the present material showed atrophy + electromyographic denervation potentials in *musculus extensor digitorum brevis* in 11 cases (14.9 %) of the 74 cases of herniation of the disk between LIV and LV in group 1.

Of the 25 cases of herniation of the disk between LV and SI KUGELBERG & PETERSEN found atrophy of the *musculus extensor digitorum brevis* in 2 and of the 60 with lumbosacral disk herniation

(group 1) in the present material 9 were found to have atrophy and denervation of the muscle distal to the lesion. This finding is discussed in further detail in the chapter on Correlation with operative findings.

#### IV *Impaired sensibility*

ROUSSET (1804) appears to have been the first to describe sensory disturbances in sciatia.

NOTTA (1854) studied these sensory disturbances in greater detail and stressed that they never involve the entire area of innervation but only irregular parts of it.

HUBERT VALLBOUX (1870) claimed that sciatia always shows sensory disturbances but PHILPIN (1895) stated that although such disturbances are common they are not constant accompaniments of sciatia. They are characterized by lack of uniformity.

DUBARRY (1902) found impaired sensibility in 50 % of his cases.

PHILPIN pointed out that in a number of cases in which the anesthesia coincided with the spread of one or more nervous branches the underlying muscles innervated by the same nerve often atrophied.

LORTAT JACOB & SABARLANU (1904) showed that some cases of sciatia were accompanied by sensory disturbances of radicular distribution and suggested the term *sciatique radiculaire* as a particular form of the disease.

Many authors claim that this form of sciatia is very common (DJIRINE, WERTHEIM, SALOMONSON, IFFRI & SCHAFFER, STURBERG).

According to HELLWIG (1920) those cases in which sensory disturbances occurred the latter are localized to the area of supply of the larger or smaller cutaneous nerves and never to the area of the plexus or the roots.

In 1931 STRANSTRÖM demonstrated the occurrence of "sensory disturbances" in 26 of 110 patients and showed that their spread coincided with the skin segments of the 1st and 2nd sacral root.

SAHLGREN in his series of 53 cases of sciatica (unoperated) found 21 to have sensory disturbances with radicular distribution corresponding to the 5th lumbar and 1st sacral root

In cases of sciatica due to disk herniation sensory disturbances have been described with varying frequency within the dermatome of the 5th lumbar and 1st sacral root (FINCHER 84 % BRADFORD & SPURLING 77 % LOVE & WALSH 21 % BARR & MIXTER 35 %) PENNYBACKER found sensory disorders in 21 cases out of 28 It was above all SPURLING & BRADFORD (1939) who pointed out the significance of the spread of impaired sensibility in the location of a lumbar disk herniation and they claimed that knowledge of the extent of impaired sensibility was often enough to locate the level of the herniation

MALMIROS (1942) on the other hand expressed the view that the level of the lesion cannot be determined on the basis of sensory disturbances

NORLEN (1944) found that it was difficult to outline the limits of the dermatome on clinical grounds only NORLEN arrived at the conclusion that if a patient with sciatica has impaired sensibility of the great toe or of the adjacent parts of the dorsum of the foot it suggests disk herniation between LIV and LV

#### *Sensory disturbances*

According to different authors

|                               |    |   |   |
|-------------------------------|----|---|---|
| 1940 LOVE & WALSH             | 21 | ♂ |   |
| 1941 BARR & MIXTER            | 35 | ♂ | (of 139)  |
| 1941 CRAIG & WALSH            | 57 | ♂ | (285/500)   |
| 1941 IRIBERG                  | 64 | ♂ | ( 8/44)   |
| 1942 MALMIROS                 | 58 | ♂ | (52/90)   |
| 1944 NORLEN                   | 64 | ♂ | (63/99) on the basis of the subjective symptoms objective findings in only 41 cases |
| 1947 AITKEN & BRADFORD        | 59 | ♂ | (18/82) disk herniations  |
| 1947 —                        | 44 | ♂ | (27/62) negative explorations   |
| 1948 WARIS                    | 62 | ♂ | (224/374)   |
| 1949 RÖVIG                    | 6  | ♂ | (16/100)  |
| 1961 Author's group 1         | 29 | ♂ | (53/182)  |
| 1961 Author's entire material | 29 | ♂ | (61/206)  |

KRECAN (1943) described distinct tracks of impaired sensibility in relation to the different nerve roots

IV root medial side of knee medial side of tibia to great toe

LV root lateral side of knee antero lateral part of lower leg, dorsum of foot and the 3 middle toes lateral side of heel

SI root lateral side of leg and foot out into the small toe

Other authors were unable to confirm the exact limits and extent of these tracks of impaired sensibility

JOERSTERS investigation of the skin dermatomes of the lumbar roots are cited for comparison

Das 2. Lumbaldermatom nimmt ebenfalls die Vorderseite des Unterschenkels ein den gesamten Fussrücken und in der Plantarseite die mediale Hälfte der Fusssohle sowie die 1. 2. und 3. Zehe

Das erste Sakraldermatom nimmt die gesamte Planta pedis ein und erstreckt sich von hier an der Hinterseite Aussen und Innenseite des Unterschenkels empor es greift auf die Dorsal-seite der Zehen über "

NORLÉN (1944) found impaired sensibility in 41 of his 99 patients. In patients with herniation between IV and LV NORLÉN found hypoaesthesia particularly of the dorsum of the foot or in the big toe. In patients with herniation between LV and SI he found hypoaesthesia particularly on the lateral side of the foot and the posterior of the calf

In the present material impaired sensibility was noted as follows

#### ed ty A Group 1

a) *Disk herniation between IV and SI* of the 60 patients with lumbosacral disk herniations 17 had impaired sensibility. Of these the impairment was localized to the lateral aspect of the lower leg and/or foot in 10 to the dorsum of the foot and/or the lower leg in 4 to the heel in 1 to the dorsal part of the calf in 1 and to the dorsum of the foot and great toe in 1

b) *Disk herniation between LV and LVI* Herniation at this

level was seen in 1 patient and then it was associated with impaired sensibility of the dorsum of the foot

c) *Disk herniation between LIV and LV* There were 74 patients of whom 21 had impaired sensibility of the dorsum of the foot and of the great toe (6) of the dorsum of the foot and/or lower leg (5) of the lateral aspect of the lower leg and/or of the foot (9) and on the dorsal aspect of the calf (1)

d) *Disk herniation between LIII and LIV* This group consisted of 2 patients in 1 of whom impaired sensibility was noted and then on the medial side of the lower leg

e) *Protrusion between LV and SI* This lesion was noted in 11 patients and with impaired sensibility of the lateral side of the lower leg in 2 of them

f) *Protrusion between LIV and LV* This group consisted of 14 patients in whom impaired sensibility was noted of the lateral aspect of the lower leg and/or foot in 2 and of the foot and/or lower leg in 2

g) *Negative findings* This group comprised 20 patients with impaired sensibility in 7. In 3 the impairment was localized to the lateral side of the lower leg and/or foot to the dorsum of the foot and/or leg in 3 and to the dorsum of the foot and great toe in 1

## B Group 2

a) *Disk herniation between LV and SI* —

b) *Disk herniation between LIV and LV* Of the 4 patients with disk herniation between LIV and LV 3 had impaired sensibility 2 of them of the lateral aspect of the lower leg and/or foot and 1 of them of the medial side of the foot

c) *Disk herniation between LIII and LIV* Herniation at this level was seen in 1 patient and he had impaired sensibility of the lateral side of the foot

d) *Protrusion between LV and SI* Of 3 patients in this group 1 had impaired sensibility and then of the medial side of the lower leg

e) *Protrusion between LIV and LV* —

f) *Protrusion between LIII and LIV* —

g) *Adhesions between LIV and SI* There were 3 patients with such adhesions and 2 of them had impaired sensibility of the lateral side of the lower leg and/or foot

h) *Adhesions between LIV and LV* —

i) *Adhesions between LIV and LV and between LV and SI* 2 patients showed such adhesions and 1 of them had impaired sensibility of the lateral side of the foot

j) *Negative findings* —

### V Muscular atrophy

As early as 1764 COTUGNO observed that patients with severe sciatica had pronounced muscular atrophy and he claimed that this atrophy was due to compression of the nerve by increasing oedema of the nerve sheath

VALLEIX (1841) expressed the view that such advanced muscular atrophy was very rare and that it occurred mainly in the serious cases but he also observed that muscular atrophy was sometimes missing in severe cases but present in moderate cases

LANDOUZY (1876) and IERNEY (1878) distinguished 2 types of sciatica. Cases without atrophy were termed sciatic neuralgias. If muscular atrophy was present it was regarded as a sign of the presence of true neuritis

LANDOUZY described the atrophy as diffuse and involving all muscles innervated by the sciatic nerve

PETIT DUTAILLIS & DE SLIZ found 24 of their 36 patients to have hypotension in association with atrophy (in 15 cases of the gluteal musculature, in 16 of the thigh and in 11 of the calf). In their opinion sciatica due to disk herniation did not as a rule cause motor symptoms

In their review of a series AITKEN & BRADFORD (1917) found atrophy of the leg in 57 % (55 of 97)

WARIS (1948) who measured the circumference of the middle of the thigh and the largest circumference of the calf found atrophy in 31 % (47/153) of patients with lumbosacral disk herniation. The corresponding figures for disk herniation between LIV and LV were 26 % (32/129)

RÖVIG (1949) found atrophy in as many as 87 % while HANRAETZ (1959) found atrophy in only 4 %

The occurrence of muscular atrophy in the present material is listed below

### A Group 1

a) *Disk herniation between LV and SI* This group consisted of 60 patients of whom 22 had muscular atrophy 9 of the thigh and calf 7 of the calf only and 6 of the thigh only

b) *Disk herniation between LV and LVI*

c) *Disk herniation between LIV and LV* Of the 74 patients in this group 30 had muscular atrophy 14 of both the thigh and the calf 8 of the calf only and 8 of the thigh only

d) *Disk herniation between LIII and LIV* There were 2 patients in this group and 1 of them had atrophy of the thigh

e) *Protrusion between LV and SI* Of the 11 patients in this group 5 had muscular atrophy 2 of the calf only and 3 of the thigh only

f) *Protrusion between LIV and LV* This group consisted of 14 patients 2 of them had atrophy of the calf only and 2 of the thigh only

g) *Negative findings* Of the 20 patients in this group 5 had atrophy of the thigh and calf 1 of the calf only and 4 of the thigh only

### B Group 2

a) *Disk herniation between LV and SI* Of the 6 patients in this group 1 had atrophy of the thigh and calf 1 of the thigh only and 3 of the calf only

b) *Disk herniation between LIV and LV* Of the 4 patients in this group 1 had atrophy of the thigh and calf

c) *Disk herniation between LIII and LIV*

d) *Protrusion between LV and SI* There were 3 patients with this lesion and 1 of them had atrophy of the thigh

e) *Protrusion between LIV and LV* —

f) *Protrusion between LIII and LIV* —



g) *Adhesions between LV and SI* There was 1 patient with atrophy of the thigh and calf

h) *Adhesions between LIV and LV* Of the patients with this lesion 1 had atrophy of the thigh and calf

i) *Adhesions between LV and SI+LIV and IV* 2 patients had this lesion and 1 of them had atrophy of the thigh

j) *Negative findings* —

50

## VI *Lasègue's test*

It is clear from all publications on lumbar disk herniation that Lasègue's test is of decisive importance for the clinical diagnosis of the disease

BARR & MIXTER (1941) in a series of 135 patients with lumbar disk herniation usually found Lasègue's sign positive LOVE & WALSH (1940) found Lasègue to be positive in 84 % (480 cases), and CRAIG & WALSH (1941) have similar figures namely 81 % (280 cases)

FRIBERG (1941) found Lasègue's sign to be positive on one side in 34 % and on both sides in 59 % of his 44 cases

RÖVIG (1949), whose series consisted of 100 cases found Lasègue's sign to be positive in 98 %

HANRAETZ (1959) found in a series from Ursula Clinic (number of cases not stated) that Lasègue's sign was positive in herniation of the lowermost disks in 94 % (also crossed in 46 %) and in herniation of the disk between LIV and LV in 60 %

In the present material the results of Lasègue's test were as follows in Table 9

## Myelographic findings

All myelographic examinations were performed with water soluble contrast medium (Abrodil Contrast L) which was first used for myelography by ARVILL & LIDSTRÖM (1931) The method employed in the present investigation was that described by LINDBLÖM (1946)

193 of the examinations were performed at the Roentgen diagnostic Department H University Hospital of Lund and the remaining 13 in other hospitals but the films taken in these

TABLE 9  
*Lasegue's test*

| Group 1            | Negative | Positive | Bilateral | Crossed |
|--------------------|----------|----------|-----------|---------|
| Herniation L V—S I | 2        | 58       | 26        | 14      |
| L V—L VI           | —        | 1        | —         | —       |
| L IV—L V           | 3        | 41       | 39        | 19      |
| L III—L IV         | —        | 2        | —         | —       |
| Protrusion L V—S I | —        | 11       | 9         | 4       |
| L IV—L V           | 2        | 12       | 9         | 3       |
| Negative findings  | 2        | 18       | 9         | 1       |
| Group 2            | 9        | 22       | 10        | 2       |
| Total              | 11       | 195      | 102       | 43      |

Total positive 94.7 %

Crossed 20.9 %

Unilateral 45.1 %

Total negative 5.3 %

Bilateral 49.5 %

last mentioned 13 cases were re examined at the Roentgen diagnostic Department II Lund before operation

In the evaluation of the cases consideration was given mainly to the preoperative reports but all films were checked for the present investigation in association with Dr STEN CRONQUIST of the Department of Neurologic Roentgenology Lund and sometimes also with Associate professor NORMAN

The primary roentgenologic findings and the operative findings are summarized in Tables 10 and 11

It is clear from Table 10 that in the 60 cases in which operation revealed disk herniation between L V and S I the herniation had been diagnosed by myelography at the same level in 37 (61.7 %) cases In 14 (23.3 %) of these 60 cases myelography revealed no signs of a pathological condition and in 9 (15 %) the myelogram was misleading (the misleading myelograms will be discussed in the chapter Correlation with operative findings )

In the 74 cases in which operation revealed disk herniation between L IV and L V the herniation had been diagnosed by myelography in 69 (93.2 %) while it had failed to show herniation in 1 (1.4 %) and was misleading in 4 (5.4 %)

As to disk herniation between L III and L IV (2) and L V and

TABLE 10  
Group 1

| Operative finding     | Myelography positive |        |        |          |          |                  |                           | Agel. negative | Uncertain      | Only thick<br>L V-root | Only thick<br>L IV-root | Technical<br>misshap |
|-----------------------|----------------------|--------|--------|----------|----------|------------------|---------------------------|----------------|----------------|------------------------|-------------------------|----------------------|
|                       | LV-SI                | LV-LVI | LIV-LV | LIII-LIV | LII-LIII | LV-SI+<br>LIV-LV | I III-L IV+<br>I II-L III |                |                |                        |                         |                      |
| Dist. klermaton LV-SI | 56                   | —      | 9      | —        | —        | 1                | —                         | 14             | —              | —                      | —                       | —                    |
| LV-LVI                | 1                    | 1      | —      | —        | —        | —                | —                         | —              | —              | —                      | —                       | —                    |
| LIV-LV                | 74                   | —      | 69     | —        | 1        | —                | —                         | 1              | —              | —                      | —                       | —                    |
| LIII-LIV              | 2                    | —      | —      | 2        | —        | —                | —                         | —              | —              | —                      | —                       | —                    |
| Protrusion LV-SI      | 11                   | —      | 2      | —        | —        | —                | —                         | 4              | —              | —                      | —                       | —                    |
| LIV-LV                | 14                   | —      | 8      | —        | —        | 1                | —                         | 3              | 1 <sup>1</sup> | —                      | —                       | —                    |
| "Negative"            | 2                    | 1      | 5      | —        | —        | —                | 1                         | 8              | —              | 1                      | —                       | 1                    |
| Total                 | 182                  | 46     | 93     | 2        | 1        | 2                | 1                         | 30             | 1              | 1                      | 1                       | 2                    |

<sup>1</sup> The contrast medium diffuses up high. The quality of the roentgenogram is therefore unsatisfactory. To the left however no root pocket is demonstrable at level of lumbosacral disk. At level of fifth disk the outline is somewhat irregular but does not allow of any definite interpretation.

TABLE 11  
Group 2

| Op ratl fln l gs      | Myelography positive         |                             |                       |               | Myelography shows      |   | My I neg |
|-----------------------|------------------------------|-----------------------------|-----------------------|---------------|------------------------|---|----------|
|                       | I V-SI                       | I IV-I V                    | I III-I IV            | Postop I V-SI | Only SI-root III ken I |   |          |
| Diskherniation I V-SI | 3                            | —                           | —                     | 2             | —                      | 1 | 1        |
| I IV-I V              | —                            | 4                           | —                     | —             | —                      | — | —        |
| I III-L IV            | —                            | —                           | (Postop I + I IV-I V) | —             | —                      | — | —        |
| I protrusions I V-SI  | —                            | 1 postop or diskhernia tion | —                     | 1             | 1                      | — | —        |
| I IV-I V              | —                            | 1                           | —                     | —             | —                      | — | —        |
| I III-I IV            | —                            | —                           | 1                     | —             | —                      | — | —        |
| Adhesions L V-SI      | 1                            | —                           | —                     | —             | —                      | 2 | —        |
| I IV-I V              | —                            | 2                           | —                     | —             | —                      | — | —        |
| I V-SI+               | [1 diskhernia tion + postop] | —                           | —                     | 1             | —                      | — | —        |
| I IV-I V              | —                            | —                           | —                     | —             | —                      | — | —        |
| Negative              | —                            | —                           | —                     | —             | —                      | 1 | 1        |
| Total 24              | 5                            | 8                           | 2                     | 4             | 1                      | 4 | 4        |

LVI (1) the myelograms were correct. As to protrusion between LV and SI myelography showed a protrusion at the same level in 4 (36.4 %) at an erroneous level in 2 (18.2 %) and no herniation at all in 4 (36.4 %) while in 1 (9 %) myelography failed owing to a technical mishap.

In the examination of protrusion between LIV and IV myelography produced as in herniation at this level better results namely in 9 (64.3 %) cases of the 14 protrusions found at operation at this level myelography showed protrusion at the same level. Myelography failed to reveal a herniation in 3 (21.4 %) it was misleading in 1 (7.1 %) and uncertain in 1 (7.1 %).

Of the 162 cases in group 1 in which at operation a disk herniation or protrusion was found agreement was found between the myelographic findings and operative findings in 122 (75.3 %).

Among the 20 cases in which operation failed to show any disk changes myelography showed no signs of a pathological condition in 8 (40 %). Of the remaining 12 cases myelography failed owing to a technical mishap in 1 (5 %) and was misleading in 11 (55 %). These cases are discussed in a subsequent chapter. Correlation with operative findings.

Concerning the myelographic findings in group 2 the number of cases was small and therefore percentages will not be given.

Most authors have not compared the frequency of agreement and disagreement between the myelographic and operative findings at the various levels but such a comparison can be made from the material of IRIBER & HUIT (1951) for example. The contrast medium used in that investigation was also Abrodil. Of 121 lumbosacral herniations 96 (79.3 %) were demonstrated in the myelogram at the same level. HANRAFTZ (1959) reported myelography to have shown disk herniation at correct level in 35 (60.3 %) of 58 cases of disk herniation at the lumbosacral space. But he used Lipiodol as a contrast medium. For the space between LIV and IV IRIBER & HUIT reported agreement between the operative and myelographic findings in 116

(92.1 %) out of 126 cases and HANRAETZ in a series in 54 (83.1 %) out of 65 cases. The figures in the material of FARBERG & HULT are not quite comparable because they have excluded 19 cases.

Most authors simply state how frequently operation confirmed the myelographic findings without stating anything about the level.

Of series examined myelographically with Abrodil mention might be made of JOHANSSON's (1949) series of 111 cases in which agreement was found between the myelographic and operative findings in 90 %.

WÖRINGER THOMASKE & BAUMGARTNER (1956) reported on a series of 346 cases. In 293 (98.3 %) cases out of 298 showing myelographic changes operation confirmed the changes demonstrated by myelography (disk herniation in 93.2 % other root compression in 5.1 %) of the 39 cases in which myelography showed no signs of a pathological condition operation revealed changes in 11 (28.2 %).

DEL BONO (1957) described a series of 373 cases in which a herniated disk found at operation was preoperatively diagnosed by myelography at the same level in 82.7 %. In 9.3 % the method failed to reveal changes afterwards found at operation it showed a change but at the wrong level in 4.6 % it showed a change that could not be confirmed at operation in 1.8 % and it showed no signs of a pathological condition in 1.6 % in which operation revealed no change either. In other words good agreement was found between myelography and the operative findings in 84.3 %.

HIRSCH (1958) found positive myelograms to show changes which were confirmed at operation in 90 % and by negative myelograms he had positive operative findings in 40 %.

FASSBENDER HAUSSLER & STÜSSEL (1958) reported agreement between the myelographic and operative findings in 88.6 % of 470 cases (Myelographic changes confirmed at operation in 97 % negative myelography — positive findings at operation in 8.6 %).

KOLSTAD & SOLEM (1959) whose series consisted of 173

cases found myelography to give correct findings in 100 (89.5 %) uncertain in 6 (3.5 %) it failed to show the lesion in 7 (4.1 %) and the findings could not be confirmed at operation in 5 (2.9 %)

Of series in which myelography was performed with Panto-  
paque mention might be made of FORD & KEY (1950) (206 cases) in which agreement was found between the myelographic and operative findings in 72.3 % FORD RAMSEY HOLT & KEY (1952) (100 cases) in which agreement was found in 60 % and finally LANSCH & FORD (1960) (560 cases) in which the operative findings were in complete accord with the myelographic findings in 453 cases giving an accuracy of 80.9 %

As to the present material agreement was found between the operative findings and the examination findings in 71.4 % in group 1 and in 72.8 % of the entire present material. The corresponding figure for FRIBERG & HULT's series was 82.1 %

On check examination of the roentgenograms interest was focused particularly on 1) thickened nerve roots 2) filling defects of nerve sheaths 3) dislocated nerve roots. All of these changes have been discussed previously and described by NICHOLS & NOSIC (1940) BUNTS (1941) ICKSELL (1943) ARNELL (1944) STRANSTRÖM (1948) JOHANSSON (1949) ICKELTSON (1951) WALK (1958) and DAUM SMITH WALKER CHAI MAN & LIVERSMAN (1959) and of course they partly were noted even in the preoperative reports

On careful examination for these 3 factors 9 of the 14 myelograms originally regarded as normal were found to show disk herniation between IV and SI at the level of the change found at operation. The percentage of agreement between the operative finding of a disk herniation between IV and SI and the myelographic findings was thereby increased from 61.7 % to 76.7 %. The corresponding figures for disk herniation between LIV and IV were 93.2 % to 94.6 % for protrusion between IV and SI from 36.4 % to 51.5 % and for protrusion between LIV and IV from 64.3 % to 71.4 %. Thus increased the frequency of agreement between the positive operative findings and myelographic findings in group 1 from 70.3 % to 83.3 %

The corresponding increase in agreement between all operative findings and myelographic findings for group 1 was from 71.4 % to 78.6 % and for the entire material from 72.8 % to 79.6 %

The frequency of thickened roots filling defects of the root sheaths and dislocated roots in relation to the operative findings is accounted for below

### Myelogram Check examination

#### A Group 1

a) *Disk herniation between LV and SI* Of the 60 cases of lumbosacral disk herniation the SI root was found to be thickened in 30 (In 1 of them the LV root was also thickened)

b) *Disk herniation between LV and LVI* Thickened SI root in 1 case

c) *Disk herniation between LIV and LV* Of these 74 cases of disk herniation the SI root was found to be thickened in 17 (18%) (6 of these cases also showed a thickened LV root)

d) *Disk herniation between LIII and LIV* —

e) *Protrusion between LV and SI* Thickened SI root in 3 of the 11 cases (1 of the cases also had a thickened LV root)

f) *Protrusion between LIV and LV* Of these 14 cases 1 had a thickened SI root (in this case also a thickened LV root)

g) *Negative operative findings* Of these 20 cases the SI root was found to be thickened in 2

#### A Group 1

a) *Disk herniation between LV and SI* 44 cases (also LV in 4 cases and LIV and LV in 1 case)

b) *Disk herniation between LV and LVI* Defect of root sheath SI in 1 case

c) *Disk herniation between LIV and LV* 9 cases (also LV in 8 cases)

d) *Disk herniation between LIII and LIV* —

e) *Protrusion between LV and SI* 6 cases (also LV in 1 case)

f) *Protrusion between LIV and LV* 2 cases (also LV in these 2 cases)

g) *Negative operative findings* 2 cases



| Dislocated A Group 1 |   |
|----------------------|---|
| SI root              |   |
| 13/60 25%            | a) Disk herniation between IV and SI 15 cases (also SI in 2 cases) (In 5 cases only SI) |
| 1/1 100%             | b) Disk herniation between IV and IVI 1 case  |
| 21/74 30%            | c) Disk herniation between LIV and LV 27 cases (also IV in 10 cases)                    |
| —                    | d) Disk herniation between LIII and LIV —   |
| 4/11 36.4%           | e) Protrusion between IV and SI 4 cases   |
| 1/14 7.1%            | f) Protrusion between IV and IV 1 case (also LV)  |
| 1/20 5%              | g) Negative operative findings 1 case   |

| Thickened A Group 1 |   |
|---------------------|---|
| LV root             |   |
| 3/60 5%             | a) Disk herniation between IV and SI 3 cases (also SI in 1 case)    |
| —                   | b) Disk herniation between IV and IVI —                             |
| 24/74 32.4%         | c) Disk herniation between LIV and LV 24 cases (also SI in 6 cases) |
| —                   | d) Disk herniation between LIII and LIV —                           |
| 2/11 18.2%          | e) Protrusion between IV and SI 2 cases (also SI in 1 case)         |
| 3/14 21.4%          | f) Protrusion between LIV and LV 3 cases (also SI in 1 case)        |
| 1/10 10%            | g) Negative findings 1 case   |

| Filling defect of root sheath IV A Group 1 |  |
|--|--|
| IV root                                    |  |
| 9/81/60 11%                                | a) Disk herniation between IV and SI 9 (8) cases (also SI in 4 cases and LIV and SI in 1 case) |
| —  | b) Disk herniation between IV and IVI —  |
| 68/74 91.9%                                | c) Disk herniation between LIV and LV 68 cases (also SI in 8 cases and LIV in 1 case)          |
| —  | d) Disk herniation between LIII and LIV —  |
| 3/11 27.3%                                 | e) Protrusion between IV and SI 3 cases (also SI in 1 case)                                    |
| 10/14 71.4%                                | f) Protrusion between LIV and LV 10 cases (also SI in 2 cases)                                 |
| 4/10 40%                                   | g) Negative findings 4 cases   |

| Dislocated A Group 1 |   |
|----------------------|---|
| LV root              |   |
| —                    | a) Disk herniation between IV and SI —  |
| —                    | b) Disk herniation between IV and IVI — |

- c) *Disk herniation between LIV and LV* 17 cases (also SI in 10 cases and LIV in 1 case)
- d) *Disk herniation between LIII and LIV* 1 case (also LIV)
- e) *Protrusion between LV and SI* —
- f) *Protrusion between LIV and LV* 2 cases (also SI in 1 case)
- g) *Negative findings* —

#### A Group 1

- a) *Disk herniation between LV and SI* —
- b) *Disk herniation between LV and LVI* —
- c) *Disk herniation between LIV and LV* —
- d) *Disk herniation between LIII and LIV* —
- e) *Protrusion between LV and SI* —
- f) *Protrusion between LIV and LV* —
- g) *Negative findings* —

#### A Group 1

- a) *Disk herniation between LV and SI* 1 case (also LIV and LV)
- b) *Disk herniation between LV and LVI* —
- c) *Disk herniation between LIV and LV* 1 case (also LV)
- d) *Disk herniation between LIII and LIV* 1 case
- e) *Protrusion between LV and SI* —
- f) *Protrusion between LIV and LV* —
- g) *Negative findings* 1 case

#### A Group 1

- a) *Disk herniation between LV and SI* —
- b) *Disk herniation between LV and LVI* —
- c) *Disk herniation between LIV and LV* 1 case (also LV)
- d) *Disk herniation between LIII and LIV* 1 case (also LV)
- e) *Protrusion between LV and SI* —
- f) *Protrusion between LIV and LV* —
- g) *Negative findings* —

#### B Group 2

- a) *Disk herniation between LV and SI* 3 cases
- b) *Disk herniation between LIV and LV* —

|                                     |  |
|-------------------------------------|--|
| —                                   | c) Disk herniation between LIII and LIV —                        |
| —                                   | d) Protrusion between LV and SI —                                |
| —                                   | e) Protrusion between IIV and LV —                               |
| —                                   | f) Protrusion between LIII and LIV —                             |
| —                                   | g) Adhesions between IV and SI —                                 |
| —                                   | h) Adhesions between LIV and IV —                                |
| 1/2                                 | i) Adhesions between LV and SI+IIV and IV 1 case                 |
| —                                   | j) Negative findings —   |
| Fulling defect of<br>root sheath SI | B Group 2  |
| 4/6                                 | a) Disk herniation between LV and SI 4 cases (also LV in 1 case) |
| 2/4                                 | b) Disk herniation between LIV and LV 2 cases (also LV)          |
| 1/1                                 | c) Disk herniation between LIII and LIV 1 case (also IIV and IV) |
| 2/3                                 | d) Protrusion between IV and SI 2 cases (also LV)                |
| —                                   | e) Protrusion between LIV and IV —                               |
| —                                   | f) Protrusion between LIII and LIV —                             |
| —                                   | g) Adhesions between IV and SI —                                 |
| —                                   | h) Adhesions between IIV and IV —                                |
| 1/2                                 | i) Adhesions between IV and SI+IIV and IV 1 case (also IV)       |
| —                                   | j) Negative findings —   |
| Dislocated<br>SI root               | B Group 2  |
| 2 0                                 | a) Disk herniation between IV and SI 2 cases                     |
| —                                   | b) Disk herniation between IIV and IV —                          |
| —                                   | c) Disk herniation between LIII and IIV —                        |
| —                                   | d) Protrusion between IV and SI —                                |
| —                                   | e) Protrusion between IIV and IV —                               |
| —                                   | f) Protrusion between LIII and IIV —                             |
| —                                   | g) Adhesions between IV and SI (dislocated SI in 1 case)         |
| —                                   | h) Adhesions between IIV and IV —                                |
| —                                   | i) Adhesions between IV and SI+IIV and IV —                      |
| —                                   | j) Negative findings —   |
| Thickening<br>IV root               | B Group 2  |
| —                                   | a) Disk herniation between IV and SI —                           |

- b) *Disk herniation between LIV and LV* —
- c) *Disk herniation between LIII and LIV* —
- d) *Protrusion between LV and SI* 1 case
- e) *Protrusion between LIV and LV* —
- f) *Protrusion between LIII and LIV* —
- g) *Adhesions between LV and SI* —
- h) *Adhesions between LIV and LV* —
- i) *Adhesions between LV and SI+LIV and LV* —
- j) *Negative findings* —

## B Group 2

- a) *Disk herniation between LV and SI* 1 case (also SI)
- b) *Disk herniation between LIV and LV* 4 cases (also SI in 2 cases)
- c) *Disk herniation between LIII and LIV* 1 case [also LIV and (SI)]
- d) *Protrusion between LV and SI* 2 cases (also SI)
- e) *Protrusion between LIV and LV* —
- f) *Protrusion between LIII and LIV* (1) case (also LIV)
- g) *Adhesions between LV and SI* 1 case
- h) *Adhesions between LIV and LV* 2 cases
- i) *Adhesions between LV and SI+LIV and LV* 1 case (also SI)
- j) *Negative findings* —

## B Group 2

- a) *Disk herniation between LV and SI* —
- b) *Disk herniation between LIV and LV* 1 case
- c) *Disk herniation between LIII and LIV* —
- d) *Protrusion between LV and SI* —
- e) *Protrusion between LIV and LV* —
- f) *Protrusion between LIII and LIV* —
- g) *Adhesions between LV and SI* —
- h) *Adhesions between LIV and LV* —
- i) *Adhesions between LV and SI+LIV and LV* —
- j) *Negative findings* —

|   |   |
|---|---|
| —                                       | c) <i>Disk herniation between LIII and LIV</i> —                        |
| —                                       | d) <i>Protrusion between LV and SI</i> —                                |
| —                                       | e) <i>Protrusion between LIV and LV</i> —                               |
| —                                       | f) <i>Protrusion between LIII and LIV</i> —                             |
| —                                       | g) <i>Adhesions between IV and SI</i> —                                 |
| —                                       | h) <i>Adhesions between LIV and IV</i> —                                |
| 1/2                                     | i) <i>Adhesions between LV and SI+IV and IV</i> 1 case                  |
| —                                       | j) <i>Negative findings</i> —   |
| <i>Filling defect of root sheath SI</i> | B Group 2   |
| 4/6                                     | a) <i>Disk herniation between LV and SI</i> 4 cases (also LV in 1 case) |
| 2/4                                     | b) <i>Disk herniation between LIV and LV</i> 2 cases (also LV)          |
| 1/1                                     | c) <i>Disk herniation between LIII and LIV</i> 1 case (also LIV and LV) |
| 2/3                                     | d) <i>Protrusion between IV and SI</i> 2 cases (also IV)                |
| —                                       | e) <i>Protrusion between LIV and IV</i> —                               |
| —                                       | f) <i>Protrusion between LIII and LIV</i> —                             |
| —                                       | g) <i>Adhesions between IV and SI</i> —                                 |
| —                                       | h) <i>Adhesions between IV and LV</i> —                                 |
| 1/2                                     | i) <i>Adhesions between IV and SI+LIV and IV</i> 1 case (also LV)       |
| —                                       | j) <i>Negative findings</i> —   |
| <i>Dislocated SI root</i>               | B Group 2   |
| 2/6                                     | a) <i>Disk herniation between IV and SI</i> 2 cases                     |
| —                                       | b) <i>Disk herniation between LIV and IV</i> —                          |
| —                                       | c) <i>Disk herniation between LIII and LIV</i> —                        |
| —                                       | d) <i>Protrusion between IV and SI</i> —                                |
| —                                       | e) <i>Protrusion between LIV and IV</i> —                               |
| —                                       | f) <i>Protrusion between LIII and LIV</i> —                             |
| —                                       | g) <i>Adhesions between IV and SI</i> (dislocated SIH in 1 case)        |
| —                                       | h) <i>Adhesions between IV and IV</i> —                                 |
| —                                       | i) <i>Adhesions between IV and SI+IV and IV</i> —                       |
| —                                       | j) <i>Negative findings</i> —   |
| <i>Thickened IV root</i>                | B Group 2   |
| —                                       | a) <i>Disk herniation between IV and SI</i> —                           |

The myelographic findings will be discussed further in the chapter Correlation with operative findings

### Electromyographic findings

In the present investigation a DISA electromyograph and concentric needle electrodes (DISA 13K03) were used

If a normal striated muscle is tested by electromyography during rest no electric activity will be recorded. If however a skeletal muscle for some reason or other has been denervated so called denervation potentials will be recorded even from the resting muscle approximately 18—21 days after the lesion responsible for denervation. The spontaneous abnormal action potentials appear with short duration and low voltage (0.5—3 msec and 50—200  $\mu$ V). They are triphasic (the third phase is however of very small amplitude). At the same time a characteristic high pitched clicking sound is heard in the loudspeaker. In addition to these denervation potentials monophasic so called positive sharp waves may be noted for the denervated musculature. (They are of 4—8 msec duration.)

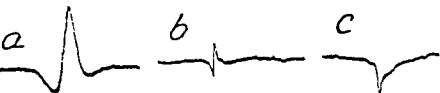


Fig 1 Motor unit potential (a) fibrillation potential (b) positive denervation potential (c)

The following sources of error must be borne in mind

a) On insertion of the electrodes or on movement of the electrodes the electromyogram may show changes which can readily be confused with denervation potentials. This phenomenon is however of very short duration.

b) If the needle electrode happens to be inserted into one of the many fine nerve fibres extending through the muscle false denervation potentials may be recorded. In such cases however the patient experiences severe pain.

It is sometimes necessary to insert the needle electrode several times into the muscle before denervation potentials are demonstrable. In the present investigation a muscle examined by

electromyography was not said to be normal unless insertion of the needle at 36 different points had failed to show any denervation potentials. Three different electrodes were used for each muscle and each electrode was inserted at 4 different sites and at 3 different depths at every site.

The examination was performed with the patient in a comfortable resting position on the examination table. The following muscles were studied in each patient: erector spinae, gluteus max et med, tensor fasciae latae, biceps femoris, quadriceps femoris, peroneus longus, extensor dig long, extensor hallucis long, tibialis ant, soleus, the medial head of the gastrocnemius, the lateral head of the gastrocnemius and extensor dig brev. The electromyographic examination was performed independently of and without previous knowledge of the clinical and myelographic examination. In other words, the examiner was not influenced by knowledge of earlier diagnostic findings.

Electromyographic localization of nerve root injury is based on the finding of denervation potentials in those muscles innervated by this particular nerve root and in no other muscles. In other words, even if an isolated muscle may have multiple nerve root innervation, it should show denervation potentials only together with the other muscles innervated by the same injured nerve root. Such a group of muscles is called a myotome.

SHILA WOODS & WARDEN (1950) were the first to report the value of EMG as a diagnostic tool in the investigation of lumbar nerve root compression syndrome. Their report is based on 60 cases of lumbar nerve root compression syndrome. The operative findings in their material were as follows: 1 IV (tumour), 1 IV, 23 SI, 35 and negative operative findings: 1. In 50 of the cases, myelography (ethyl iodophenylundecylate) was performed. They compared the diagnostic possibilities of both electromyography and myelography and found the results of electromyography to coincide with those at operation in 90% (54/60) and of myelography with 87.5% (48/55).

In 1953 MARINACCI in his book "Clinical Electromyography"

reported on 150 operated lumbar disk herniations which he examined electromyographically before operation. He gave the following results:

71 cases had disk herniation between LV and SI

|             |                    |                            |
|-------------|--------------------|----------------------------|
| EMG showed  | SI                 | 62 cases                   |
|             | LV                 | 3                          |
|             | Normal             | 1 case (less than 14 days) |
| Myelography | LV — SI            | 31 cases                   |
|             | LIV — LV           | 4                          |
|             | LV — SI + LIV — LV | 2                          |
|             | Normal             | 26                         |
|             | No myelography     | 8                          |

57 of them had disk herniation between LIV and LV

|             |                    |                       |
|-------------|--------------------|-----------------------|
| EMG showed  | LV                 | 48 cases              |
|             | LV + SI            | 5                     |
|             | SI                 | 2                     |
|             | LIV                | 1 case                |
|             | Normal             | 1 (less than 14 days) |
| Myelography | LIV — LV           | 38 cases              |
|             | LIV — LV + LV — SI | 4                     |
|             | LIII — LIV         | 2                     |
|             | Opposite side      | 3                     |
|             | No myelography     | 1 case                |

15 of them had disk herniation between LIV — LV + LV — SI

|             |                    |         |
|-------------|--------------------|---------|
| EMG showed  | LV                 | 6 cases |
|             | SI                 | 4       |
|             | LV + SI            | 5       |
| Myelography | LIV — LV           | 6 cases |
|             | LV — SI            | 14      |
|             | LIV — LV + LV — SI | 2       |
|             | Normal             | 2       |
|             | No myelography     | 1 case  |

3 of them had disk herniation between LIII and LIV

|             |                       |         |
|-------------|-----------------------|---------|
| EMG showed  | LIV + LV              | 2 cases |
|             | LIII + LIV            | 1 case  |
| Myelography | LIII — LIV            | 2 cases |
|             | LIII — LIV + LIV — LV | 1 case  |

2 of them had spondylolisthesis of LV

|             |            |         |
|-------------|------------|---------|
| EMG showed  | LV + SI    | 2 cases |
| Myelography | LIII — LIV | 1 case  |
|             | Normal     | 1       |



electromyography was not said to be normal unless insertion of the needle at 36 different points had failed to show any denervation potentials. Three different electrodes were used for each muscle and each electrode was inserted at 4 different sites and at 3 different depths at every site.

The examination was performed with the patient in a comfortable resting position on the examination table. The following muscles were studied in each patient: erector spinae, gluteus max et med, tensor fasciae lata, biceps femoris, quadriceps femoris, peroneus longus, extensor dig long, extensor hallucis long, tibialis ant, soleus, the medial head of the gastrocnemius, the lateral head of the gastrocnemius and extensor dig brev. The electromyographic examination was performed independently of and without previous knowledge of the clinical and myelographic examination. In other words, the examiner was not influenced by knowledge of earlier diagnostic findings.

Electromyographic localization of nerve root injury is based on the finding of denervation potentials in those muscles innervated by this particular nerve root and in no other muscles. In other words, even if an isolated muscle may have multiple nerve root innervation, it should show denervation potentials only together with the other muscles innervated by the same injured nerve root. Such a group of muscles is called a myotome.

SHIFA WOODS & WERDER (1950) were the first to report the value of EMG as a diagnostic tool in the investigation of lumbar nerve root compression syndrome. Their report is based on 60 cases of lumbar nerve root compression syndrome. The operative findings in their material were as follows: IV (tumour) 1, IV 23, SI 35, and negative operative findings 1. In 50 of the cases, myelography (ethyl iodophenylundecylate) was performed. They compared the diagnostic possibilities of both electromyography and myelography and found the results of electromyography to coincide with those at operation in 90% (54/60) and of myelography with 87.3% (48/55).

In 1955 MARINACCI in his book "Clinical Electromyography"

reported on 150 operated lumbar disk herniations which he examined electromyographically before operation. He gave the following results:

71 cases had disk herniation between LV and SI

|             |                |                            |
|-------------|----------------|----------------------------|
| EMG showed  | SI             | 62 cases                   |
|             | LV             | 5                          |
|             | Normal         | 1 case (less than 14 days) |
| Myelography | LV—SI          | 31 cases                   |
|             | LIV—LV         | 4                          |
|             | LV—SI+LIV—LV   | 2                          |
|             | Normal         | 26                         |
|             | No myelography | 8                          |

57 of them had disk herniation between LIV and LV

|             |                |                       |
|-------------|----------------|-----------------------|
| EMG showed  | LV             | 48 cases              |
|             | LV+SI          | 5                     |
|             | SI             | 2                     |
|             | LIV            | 1 case                |
|             | Normal         | 1 (less than 14 days) |
| Myelography | LIV—LV         | 38 cases              |
|             | LIV—LV+LV—SI   | 4                     |
|             | LIII—LIV       | 2                     |
|             | Opposite side  | 3                     |
|             | No myelography | 1 case                |

15 of them had disk herniation between LIV—LV+LV—SI

|             |                |         |
|-------------|----------------|---------|
| EMG showed  | LV             | 6 cases |
|             | SI             | 4       |
|             | LV+SI          | 5       |
| Myelography | LIV—LV         | 6 cases |
|             | LV—SI          | 14      |
|             | LIV—LV+LV—SI   | 2       |
|             | Normal         | 2       |
|             | No myelography | 1 case  |

3 of them had disk herniation between LIII and LIV

|             |                 |         |
|-------------|-----------------|---------|
| EMG showed  | LIV+LV          | 2 cases |
|             | LIII+LIV        | 1 case  |
| Myelography | LIII—LIV        | 2 cases |
|             | LIII—LIV+LIV—LV | 1 case  |

2 of them had spondylolisthesis of LV

|             |          |         |
|-------------|----------|---------|
| EMG showed  | LV+SI    | 2 cases |
| Myelography | LIII—LIV | 1 case  |
|             | Normal   | 1       |

2 patients had cauda equina tumours. I MG and myelography showed the same changes which were confirmed at operation in both cases.

SHRA & WOODS (1956) described 131 cases of spinal nerve root compression studied electromyographically and myelographically (Pantopaque in most cases) and in all cases operation was done later. The results are given below.

| Root lesion found at operation | I MG           |           | Myelography    |           |
|--------------------------------|----------------|-----------|----------------|-----------|
|                                | Correct        | Incorrect | Correct        | Incorrect |
| I III—I IV (tumour)            | 1              | 0         | 1              | 0         |
| I IV—L V                       | 152            | 9         | 104            | 37        |
| I V—S I                        | 171            | 20        | 124            | 21        |
| No lesion                      | 1              | 0         | —              | —         |
| Total                          | 325<br>(91.8%) | 29        | 229<br>(79.8%) | 58        |
|                                | 354            |           | 287            |           |

The series hitherto referred to thus showed very good agreement regarding the electromyographic findings. In 1957 however CRUIKSHANK & SHIELDS described a material in which the results were not quite so good. They gave the following analysis of their 136 cases.

|                      | Large stenosis or protrusion |       | Small protrusion     |       |
|----------------------|------------------------------|-------|----------------------|-------|
|                      | Correct localization         |       | Correct localization |       |
| I V—S I<br>86 cases  | 69                           | cases | 18                   | cases |
| Clinical             | 57                           | 84    | 15                   | 82    |
| I MG                 | 56                           | 82    | 14                   | 7     |
| Myel                 | 53                           | 78    | 12                   | 6     |
| I IV—I V<br>50 cases | 34                           | cases | 16                   | cases |
| Clinical             | 22                           | 65    | 7                    | 44    |
| I MG                 | 23                           | 68    | 6                    | 31    |
| Myel                 | 30                           | 88    | 13                   | 81    |

Other authors who discussed electromyography as a diagnostic tool in the investigation of disk herniations are BOWSER & SCHMIDT (1957) who in 30 patients found an accuracy for electromyographic findings of 80 % and MENDELSON & SOLA (1958) which in 45 cases found a correct electromyography in 89 % incorrect in 7 % and normal in 4 %. In their material the myelograms were correct in 84 % incorrect in 9 % and normal in 7 %. GOLSETH (1950) VON HAGEN (1953) only described the results in small materials (some single cases) and will therefore not be discussed further here.

Among these earlier investigators MARINACCI gave the following distribution of myotomes:

| Muscle   | Myotome |
|--|---------|
| Quadriceps femoris<br>Tibialis anterior<br>Erector spinae at the level of LIV—LV vertebrae   | LIV     |
| Tibialis anterior<br>Ext. hall. and dig. long.<br>Lateral head of gastrocnemius<br>The peroneal group<br>Hamstrings<br>Tensor fascia lata<br>Gluteus med.<br>Erector spinae at the level of the lumbosacral nerves | LV      |
| Medial head of gastrocnemius<br>Soleus<br>Hamstrings<br>Gluteus max.<br>Erector spinae at the level of SI—SII vertebrae<br>The peroneal group  | SI      |

SHEA, WOODS & WERDEN and SHEA & WOODS used largely the same classification but did not test the erector spinae. CRUE, PUTENZ & SHILDEN on the other hand, like MARINACCI, tested the erector spinae and also used largely the same myotome classification.

In the present investigation this schema was also adopted.

2 patients had cauda equina tumours. I MG and myelography showed the same changes which were confirmed at operation in both cases.

SHEA & WOODS (1956) described 354 cases of spinal nerve root compression studied electromyographically and myelographically (Pantopaque in most cases) and in all cases operation was done later. The results are given below.

| Root lesion found at operation | I MG           |           | Myelography    |           |
|--------------------------------|----------------|-----------|----------------|-----------|
|                                | Correct        | Incorrect | Correct        | Incorrect |
| I III—I IV (tumour)            | 1              | 0         | 1              | 0         |
| I IV—L V                       | 152            | 9         | 101            | 37        |
| I V—S I                        | 171            | 20        | 124            | 21        |
| No lesion                      | 1              | 0         | —              | —         |
| Total                          | 325<br>(91.8%) | 29        | 226<br>(79.8%) | 58        |
|                                | 354            |           | 284            |           |

The series hitherto referred to thus showed very good agreement regarding the electromyographic findings. In 1957, however, CRUP, PUDENZ & SHIELDS described a material in which the results were not quite so good. They gave the following analysis of their 136 cases.

|                      | Large extrusion or protrusion |       | Small protrusion     |       |
|----------------------|-------------------------------|-------|----------------------|-------|
|                      | Correct localization          |       | Correct localization |       |
| I V—S I<br>86 cases  | 65                            | cases | 18                   | cases |
| Clinical             | 57                            | 81    | 15                   | 83    |
| I MG                 | 56                            | 82    | 14                   | 81    |
| Myel                 | 53                            | 79    | 12                   | 67    |
| I IV—I V<br>50 cases | 34                            | cases | 16                   | cases |
| Clinical             | 27                            | 65    | 7                    | 44    |
| I MG                 | 23                            | 68    | 6                    | 37    |
| Myel                 | 30                            | 89    | 13                   | 81    |

Other authors who discussed electromyography as a diagnostic tool in the investigation of disk herniations are BONNER & SCHMIDT (1957) who in 30 patients found an accuracy for electromyographic findings of 80 % and MENDELSON & SOLA (1958) which in 40 cases found a correct electromyography in 89 % incorrect in 7 % and normal in 4 %. In their material the myelograms were correct in 84 % incorrect in 9 % and normal in 7 %. GOLSTEN (1950) and HAGEN (1955) only described the results in small materials (some single cases) and will therefore not be discussed further here.

Among these earlier investigators MARINACCI gave the following distribution of myotomes

| Myotome   | Myotome |
|---|---------|
| Quadriceps femoris<br>Tibialis anterior<br>Erector spinae (at the level of LIV—LV vertebrae)  | I IV    |
| Tibialis anterior<br>Ext hallucis digitorum<br>Lateral head of gastrocnemius<br>The peroneal group<br>Hamstrings<br>Tensor fasciae latae<br>Gluteus medius<br>Erector spinae (at the level of the lumbosacral interspace) | I V     |
| Medial head of gastrocnemius<br>Soleus<br>Hamstrings<br>Gluteus maximus<br>Erector spinae (at the level of SI—SII vertebrae)<br>The peroneal group  | SI      |

SHEA, WOODS & WERDEN and SHEA & WOODS used largely the same classification but did not test the erector spinae. CRUE, PUDENZ & SHELDEY on the other hand like MARINACCI tested the erector spinae and also used largely the same myotome classification.

In the present investigation this schema was also adopted.

but with the following modifications. Of hamstrings only the biceps femoris was tested. Of the peroneal group only the peroneus longus and as an addition the ext dig brev (IV inner vated) was tested.

The findings made at this examination are given below.

#### on A Group 1

- a) Disk herniation between IV and SI 48 cases (including 5 with IV and 2 with IIV)
- b) Disk herniation between IV and IVI —
- c) Disk herniation between IIV and IV 20 cases (including 8 with IV)
- d) Disk herniation between III and IIV —
- e) Protrusion between IV and SI 9 cases (including 2 with IV)
- f) Protrusion between IIV and IV 5 cases (including 2 with IV)
- g) Negative findings 8 cases (including 1 with IV)

#### on A Group 1

- a) Disk herniation between IV and SI 12 cases (including 5 with SI)
- b) Disk herniation between IV and IVI 1 case
- c) Disk herniation between IIV and IV 56 cases (including 8 with SI and 1 with IIV)
- d) Disk herniation between III and IIV —
- e) Protrusion between IV and SI 2 cases (both of them with SI)
- f) Protrusion between IIV and IV 11 cases (including 1 with SI and 1 with IIV)
- g) Negative findings 7 cases (including 1 with SI and 1 with IIV)

#### on A Group 1

- a) Disk herniation between IV and SI 2 cases (both of them with SI)
- b) Disk herniation between IV and IVI —
- c) Disk herniation between IIV and IV 1 case (with IV)

- d) *Disk herniation between LIII and LIV* 2 cases
- e) *Protrusion between LV and SI* —
- f) *Protrusion between LIV and LV* —
- g) *Negative findings* 1 case (with LV)

#### B Group 2

- a) *Disk herniation between LV and SI* 6 cases (including with IV)
- b) *Disk herniation between LIV and LV* 1 case
- c) *Disk herniation between LIII and LIV* —
- d) *Protrusion between LV and SI* 2 cases
- e) *Protrusion between LIV and LV* —
- f) *Protrusion between LIII and LIV* —
- g) *Adhesions between LV and SI* 3 cases (including 1 with LV)
- h) *Adhesions between LIV and LV* —
- i) *Adhesions between LV and SI+LIV and LV* 1 case (with LV)
- j) *Negative findings* —

#### B Group 2

- a) *Disk herniation between LV and SI* 2 cases (with SI)
- b) *Disk herniation between LIV and LV* 3 cases
- c) *Disk herniation between LIII and LIV* 1 case (with LIV)
- d) *Protrusion between LV and SI* —
- e) *Protrusion between LIV and LV* 1 case
- f) *Protrusion between LIII and LIV* —
- g) *Adhesions between LV and SI* 1 case (with SI)
- h) *Adhesions between LIV and LV* 2 cases (including 1 with LIV)
- i) *Adhesions between LV and SI+LIV and LV* 2 cases (including 1 with SI)
- j) *Negative findings* —

#### B Group 2

- a) *Disk herniation between LV and SI* —
- b) *Disk herniation between LIV and LV* —



but with the following modifications. Of hamstrings only the biceps femoris was tested. Of the peroneal group only the peroneus longus and in addition the ext. dig. brev. (I V inner vided) was tested.

The findings made at this examination are given below

#### on A Group 1

- a) Disk herniation between L V and S I 48 cases (including 5 with L V and 2 with L I V)
- b) Disk herniation between I V and L V I —
- c) Disk herniation between I I V and I V 20 cases (including 8 with L V)
- d) Disk herniation between I I I and L I V —
- e) Protrusion between I V and S I 9 cases (including 2 with I V)
- f) Protrusion between I I V and I V 5 cases (including 2 with I V)
- g) Negative findings 8 cases (including 1 with I V)

#### on A Group 2

- a) Disk herniation between I V and S I 12 cases (including 5 with S I)
- b) Disk herniation between I V and I V I 1 case
- c) Disk herniation between I I V and I V 56 cases (including 8 with S I and 1 with I I V)
- d) Disk herniation between I I I and L I V —
- e) Protrusion between I V and S I 2 cases (both of them with S I)
- f) Protrusion between L I V and I V 11 cases (including 1 with S I and 1 with I I V)
- g) Negative findings 7 cases (including 1 with S I and 1 with L I V)

#### on A Group 3

- a) Disk herniation between I V and S I 2 cases (both of them with S I)
- b) Disk herniation between I V and I V I —
- c) Disk herniation between I I V and I V 1 case (with I V)

|  |                        |
|--|------------------------|
| Ca mb  |                        |
|  | H m t d d k L III—L IV |
| 1704/58<br>1077/59   |                        |
|  | P tru L V—S I          |
| 117/58<br>464/58<br>941/58<br>120/58<br>1444/58<br>631/58<br>50/59<br>1113/9<br>1309/59<br>1179/9<br>178/59  |                        |
|  | Prot L IV—L V          |
| 607/58<br>46/58<br>784/58<br>635/58<br>1173/58<br>314/58<br>158/59<br>59<br>882/59<br>96/59<br>1260/9<br>1618/9<br>1640/9<br>109/9   |                        |
|  | N d k h g              |
| 187/58<br>190/58<br>31/58<br>306/58<br>631/58<br>822/58<br>879/58<br>884/58<br>1014/58<br>1233/58<br>1459/58<br>1638/58<br>104/58<br>11/9<br>41/9<br>51/9<br>80/59<br>874/59<br>93/59<br>1733/59 |                        |

Fig 4

## Group 2

|  |  |
|--|--|
| Ca mb  |  |
| 185/58<br>160/58<br>121/58<br>101/58<br>18/58<br>316/58<br>487/58<br>497/58<br>95/58<br>974/58<br>1203/58<br>177/58<br>185/58<br>106/9<br>179/9<br>333/9<br>54/59<br>05/59<br>922/9<br>977/59<br>1527/9<br>1150/59<br>1310/59<br>1866/59 |  |

Fig 5

Black marks the occurrence of denervation potentials in the muscle

- c) *Dist herniation between LIII and LIV* 1 case (with IV)
- d) *Protrusion between IV and SI* —
- e) *Protrusion between IV and V* —
- f) *Protrusion between III and IV* 1 case
- g) *Adhesions between IV and SI* —
- h) *Adhesions between LIV and V* 1 case (with V)
- i) *Adhesions between V and SI+III and V* —
- j) *Negative findings* —

Figs 2—5 show in which muscles denervation potentials were demonstrable

The diagnostic results and the incorrect electromyograms are discussed in the next chapter

## CORRELATION OF DIFFERENT DIAGNOSTIC METHODS WITH OPERATIVE FINDINGS

It is apparent from the description of the material in chapter III that projection of pain and impaired sensibility are of relatively little value in assessing the level of a lesion. Therefore on comparison of the value of the various examination findings for assessing the level of lumbar root compression syndrome below these two factors will be disregarded. This also applies to Lasègue's test and muscular atrophy which except for atrophy of *musculus ext dig brev* is of no value in locating the level of a lesion.

As to the clinical neurological examination attention was thus given to reflex disorders and weakening or paralysis of the extensors of the great toe.

Below a comparison is made of the value of these clinical findings of the primary myelographic findings and of the electromyographic findings judged from the findings at operation. The results of the comparison are summarized in the tables below.

It is clear from Table 12 that lumbosacral disk herniation produced clinically and electromyographically demonstrable changes of SI in 80 % while myelography gave a correct positive diagnosis in only 61.7 %. This is in agreement with earlier experiences and may be explained by the relatively large diameter of the spinal canal in the lumbosacral space. Of the cases of disk herniation between LIV and LV and revealed at operation however myelography gave correct information in 93.2 % while clinical examination and electromyography suggested affection of LV in only 75.7 %.

As to protrusions best agreement was found between electro

| Operation findings                     | Clinical neurological examination |      |            |      |             |             |    |
|--|-----------------------------------|------|------------|------|-------------|-------------|----|
|  | Correct                           |      | Misleading |      | Normal      |             |    |
|  | Number                            | %    | Number     | %    | Number      | %           |    |
| Diskherniation I V—S I                 | 48                                | 80   | 10         | 16.7 | 2           | 3.3         | 3  |
| "    I IV—I V                          | 56                                | 75.7 | 10         | 13.5 | 8           | 10.8        | 0  |
| "    I III—I IV                        | 2                                 | 100  | —          | —    | —           | —           |    |
| Protrusion I V—S I                     | 8                                 | 72.8 | 2          | 18.2 | 1           | 9.1         |    |
| "    I IV—L V                          | 10                                | 71.4 | 1          | 7.1  | 3           | 21.4        |    |
| Total with positive operation findings | 124                               | 77   | 23         | 14.3 | 14          | 8.7         | 12 |
| Negative findings                      | 4                                 | 20   | 16         | 80   | See correct | See correct |    |
| Total number of cases                  | 128                               | 70.7 | 39         | 21.5 | 14 (18)     | 7.7 (9.9)   | 13 |

myelographic and operative findings 81.8 % for lumbosacral protrusions and 78.6 % for protrusions between I IV and I V while the corresponding figures for operative findings versus clinical neurological findings were 72.8 % (I V and S I) and 71.4 % (L IV and I V) respectively and for operative findings versus myelographic findings 36.4 % (I V and S I) and 61.3 % (I IV and I V) respectively.

In group 1 best agreement was found between the positive operative findings and electromyography namely 78.3 % while the corresponding figure for the clinical neurological examination was 77 % and for myelography 75.2 %.

The relatively low number of correct diagnostic results in the group in which operation revealed no signs of a pathological disk condition is due to the fact that the findings were classified as "negative" simply because they showed no disk changes. But it was only in 8 out of the 20 cases in which operation revealed no signs of a pathological condition. The other 12

## A Group 1

| Myelography |      |             |             |                  |     | Electromyography |      |             |      |             |             |
|-------------|------|-------------|-------------|------------------|-----|------------------|------|-------------|------|-------------|-------------|
| Mislead no. |      | Normal      |             | Technical mishap |     | Correct          |      | Mislead no. |      | Normal      |             |
| Number      |      | Number      |             | Number           |     | Number           |      | Number      |      | Number      |             |
| 9           | 10   | 14          | 23 3        | —                | —   | 48               | 80   | 7           | 11   | 0           | 83          |
| 4           | 54   | 1           | 14          | —                | —   | 56               | 107  | 12          | 16 2 | 6           | 81          |
| —           | —    | —           | —           | —                | —   | 2                | 100  | —           | —    | —           | —           |
| 2           | 18 2 | 4           | 36 4        | 1                | 91  | 9                | 81 8 | —           | —    | 2           | 18 7        |
| 2           | 14 3 | 3           | 21 4        | —                | —   | 11               | 18 6 | 3           | 21 4 | —           | —           |
| 17          | 10 6 | 22          | 13 7        | 1                | 0 5 | 126              | 18 3 | 20          | 13 7 | 13          | 81          |
| 10          | 50   | See correct | See correct | 1                | 5   | 6                | 30   | 14          | 70   | See correct | See correct |
| 27          | 14 9 | 22 (29)     | 12 2 (16 0) | 2                | 11  | 132              | 12 9 | 36          | 19 9 | 13 (19)     | 7 2 (10 0)  |

showed root affection due to factors other than disk involvement which is seen in Table 14

It is therefore the figures in Table 14 that should be regarded as adequate

With regard to the cases in group 2 myelography and electromyography agreed well with the findings made at operation. There was agreement in 91.7 % while the corresponding figure for the clinical neurological examination of these cases was 79.2 %

It is clear from the description of the material that in many cases combined clinical neurological findings and likewise combined electromyographic findings were made. These findings are accounted for Tables 15 to 18

The relation between unequivocal and combined clinical findings respectively and unequivocal and combined electromyographic findings respectively and the operative findings are given below

| Operation findings                     | Clinical neurological examination |      |            |      |             |              |
|--|-----------------------------------|------|------------|------|-------------|--------------|
|  | Correct                           |      | Misleading |      | Normal      |              |
|  | Number                            | %    | Number     | %    | Number      | %            |
| Diskherniation I V—S I                 | 48                                | 80   | 10         | 16.7 | 2           | 3.3          |
| " I IV—I V                             | 56                                | 75.7 | 10         | 13.5 | 8           | 10.8         |
| " I III—I IV                           | 2                                 | 100  | —          | —    | —           | —            |
| Protrusion I V—S I                     | 8                                 | 72.8 | 2          | 18.2 | 1           | 9.1          |
| " I IV—I V                             | 10                                | 71.4 | 1          | 7.1  | 3           | 21.4         |
| Total with positive operation findings | 124                               | 77   | 23         | 14.3 | 14          | 8.7          |
| "Negative findings                     | 4                                 | 20   | 16         | 80   | See correct | See correct  |
| Total number of cases                  | 128                               | 70.7 | 39         | 21.5 | 14<br>(18)  | 7.7<br>(9.9) |

myographic and operative findings 81.8 % for lumbosacral protrusions and 78.6 % for protrusions between I IV and I V while the corresponding figures for operative findings versus clinical neurological findings were 72.8 % (I V and S I) and 71.4 % (I IV and I V) respectively and for operative findings versus myelographic findings 36.4 % (I V and S I) and 61.3 % (I IV and I V) respectively.

In group 1 best agreement was found between the positive operative findings and electromyography namely 78.3 % while the corresponding figure for the clinical neurological examination was 77 % and for myelography 75.2 %.

The relatively low number of correct diagnostic results in the group in which operation revealed no signs of a pathological disk condition is due to the fact that the findings were classified as "negative" simply because they showed no disk changes. But it was only in 8 out of the 20 cases in which operation revealed no signs of a pathological condition. The other 12

## A Group 1

| Myelography |      |             |             |                  |     | Electromyography |      |            |      |             |             |
|-------------|------|-------------|-------------|------------------|-----|------------------|------|------------|------|-------------|-------------|
| Misleading  |      | Normal      |             | Technical mishap |     | Correct          |      | Misleading |      | Normal      |             |
| Number      |      | Number      |             | Number           |     | Number           |      | Number     |      | Number      |             |
| 9           | 15   | 14          | 23.3        | —                | —   | 48               | 80   | 7          | 11.7 | 5           | 8.3         |
| 4           | 5.4  | 1           | 1.4         | —                | —   | 56               | 75.7 | 19         | 16.2 | 6           | 8.1         |
| —           | —    | —           | —           | —                | —   | 2                | 100  | —          | —    | —           | —           |
| 2           | 18.2 | 4           | 36.4        | 1                | 9.1 | 9                | 81.8 | —          | —    | 2           | 18.2        |
| 2           | 14.3 | 3           | 21.4        | —                | —   | 11               | 78.6 | 3          | 21.4 | —           | —           |
| 17          | 10.6 | 22          | 13.7        | 1                | 0.5 | 126              | 18.3 | 22         | 13.7 | 13          | 8.1         |
| 10          | 50   | See correct | See correct | 1                | 5   | 6                | 30   | 14         | 10   | See correct | See correct |
|             |      | 22          | 12.2        |                  |     |                  |      |            |      | 13          | 1.2         |
| 21          | 14.9 | (79)        | (16.0)      | 2                | 1.1 | 132              | 2.9  | 36         | 19.9 | (19)        | (10.5)      |

showed root affection due to factors other than disk involvement which is seen in Table 14

It is therefore the figures in Table 14 that should be regarded as adequate

With regard to the cases in group 2 myelography and electromyography agreed well with the findings made at operation. There was agreement in 91.7 % while the corresponding figure for the clinical neurological examination of these cases was 79.2 %

It is clear from the description of the material that in many cases combined clinical neurological findings and likewise combined electromyographic findings were made. These findings are accounted for Tables 15 to 18

The relation between unequivocal and combined clinical findings respectively and unequivocal and combined electromyographic findings respectively and the operative findings are given below



TABLE 13  
B (group 2)

| Operative findings   | Clinical neuro exam |          |          | Myelography |      |          | Electroencephalography |          |          |
|----------------------|---------------------|----------|----------|-------------|------|----------|------------------------|----------|----------|
|                      | Correct             | Mild     | Normal   | Correct     | Mild | Normal   | Correct                | Mild     | Normal   |
| Displacement 1 V-SI  | 2                   | 1        | -        | 5           | -    | 1        | 6                      | -        | -        |
| " 1 V-SI             | 2                   | 2        | -        | 4           | -    | -        | 3                      | 1        | -        |
| " 1 III-SI           | 1                   | -        | -        | 1           | -    | -        | 1                      | -        | -        |
| Fractures 1 V-SI     | 3                   | -        | -        | 3           | -    | -        | 2                      | -        | 1        |
| " 1 V-SI             | 1                   | -        | -        | 1           | -    | -        | 1                      | -        | -        |
| " 1 III-SI           | 1                   | -        | -        | 1           | -    | -        | 1                      | -        | -        |
| Adhesions 1 V-SI     | 3                   | -        | 1        | 2           | -    | 1        | 3                      | -        | -        |
| " 1 V-SI             | 2                   | -        | -        | 2           | -    | -        | 2                      | -        | -        |
| " 1 V-SI+            | 2                   | -        | -        | 2           | -    | -        | 2                      | -        | -        |
| " 1 V-SI             | -                   | 1        | -        | 1           | -    | -        | 1                      | -        | -        |
| Negative exploration | -                   | -        | -        | -           | -    | -        | -                      | -        | -        |
| Total                | 19 (9.2%)           | 4 (1.7%) | 3 (1.1%) | 22 (9.7%)   | -    | 2 (8.3%) | 22 (9.7%)              | 1 (4.2%) | 1 (4.2%) |
|                      |                     |          |          |             |      | (3)      |                        |          | (2)      |

TABLE 11  
Negative explorative findings (group I)

| Osteolytic changes     | Explorative examinations |                    |             | Material group I |                    |             | Total        |                    |             |
|------------------------|--------------------------|--------------------|-------------|------------------|--------------------|-------------|--------------|--------------------|-------------|
|                        | Correct                  | Misinterpretations | Negative    | Correct          | Misinterpretations | Negative    | Correct      | Misinterpretations | Negative    |
| Negative explorations  | 2                        | 0                  | See correct | 0                | 2                  | See correct | 3            | —                  | See correct |
| Osteophytes            | 4                        | 1                  | 2           | 0                | —                  | 1           | 4            | 1                  | 1           |
| Varices                | 4                        | —                  | —           | —                | 1                  | 2           | 4            | —                  | 2           |
| Varices + narrow space | 1                        | —                  | —           | 1                | —                  | —           | 1            | —                  | —           |
| Total                  | 11 (55)                  | 7 (35)             | 2 (10%) (4) | 13 (65)          | 3 (15%) (7)        | 3 (15%) (7) | 11 (55%) (6) | 6 (30%) (6)        | 3 (15%) (6) |



TABLE 18  
Operative findings group 9

| Displacement           | Disk herniation L IV-L V | Disk herniation L III-L IV | Protrusions L V-S I | Protrusions L IV-L V | Protrusions L III-L IV | Adhesions L V-S I | Adhesions L IV-L V | Adhesions L V-S I + L IV-L V | Value | Total |
|------------------------|--------------------------|----------------------------|---------------------|----------------------|------------------------|-------------------|--------------------|------------------------------|-------|-------|
| Only one root affected |                          |                            |                     |                      |                        |                   |                    |                              |       |       |
| S I                    | 4                        | —                          | 2                   | 1                    | —                      | —                 | —                  | —                            | —     | 8     |
| I V                    | 1                        | 3                          | —                   | 1                    | —                      | —                 | 1                  | 1                            | —     | 7     |
| I IV                   | —                        | —                          | —                   | 1                    | —                      | —                 | —                  | —                            | —     | 1     |
| Two roots affected     |                          |                            |                     |                      |                        |                   |                    |                              |       |       |
| S I + I V              | 3                        | —                          | —                   | —                    | —                      | 1                 | —                  | 1                            | —     | 4     |
| S I + I IV             | —                        | —                          | —                   | —                    | —                      | —                 | —                  | —                            | —     | —     |
| I V + I IV             | —                        | —                          | —                   | —                    | 1                      | —                 | 1                  | —                            | —     | 2     |
| Three roots affected   |                          |                            |                     |                      |                        |                   |                    |                              |       |       |
| S I + I V + I IV       | —                        | —                          | —                   | —                    | —                      | —                 | —                  | —                            | —     | —     |
| No root affected       |                          |                            |                     |                      |                        |                   |                    |                              |       |       |
| 0                      | —                        | —                          | 1                   | —                    | —                      | —                 | —                  | —                            | 1     | 2     |
| Total                  | 7                        | 3                          | 1                   | 4                    | 2                      | 1                 | 2                  | 2                            | 1     | 34    |

## g.) Negative explorations

TABLE 23

| Clinical  | I MC |    |          |        | Total |
|-----------|------|----|----------|--------|-------|
|           | I V  | SI | I V + SI | Normal |       |
| I V       | —    | —  | —        | 1      | 1     |
| I V + I V | —    | —  | —        | 1      | 1     |
| I V + SI  | 1    | —  | 1        | —      | 2     |
| SI        | —    | 2  | —        | —      | 2     |
| Normal    | —    | 1  | —        | 1      | 2     |
| Total     | 1    | 3  | 1        | 3      | 8     |

## h.) Osteofytes or varices

TABLE 24

| Clinical       | I MC |    |           |        | Total |
|----------------|------|----|-----------|--------|-------|
|                | I V  | SI | I V + I V | Normal |       |
| I V            | 1    | —  | —         | —      | 1     |
| I V            | 1    | —  | —         | 1      | 2     |
| I V + I V      | 1    | —  | 1         | 1      | 3     |
| I V + I V + SI | —    | 1  | —         | —      | 1     |
| SI             | —    | 2  | —         | —      | 2     |
| I V + SI       | 1    | —  | —         | —      | 1     |
| Normal         | —    | 1  | —         | 1      | 2     |
| Total          | 4    | 4  | 1         | 3      | 12    |

## B Group 2

## a) Disk herniation between I V and SI

TABLE 25

| Clinical       | I MC |          | Total |
|----------------|------|----------|-------|
|                | SI   | SI + I V |       |
| SI             | 2    | —        | 2     |
| SI + I V       | 2    | —        | 2     |
| SI + I V + I V | —    | 1        | 1     |
| I V            | —    | 1        | 1     |
| Total          | 4    | 2        | 6     |

## b) Disk herniation between LIV and LV

TABLE 26

| Clinical | EMG |    | Total |
|----------|-----|----|-------|
|          | LV  | SI |       |
| LV       | 1   | —  | 1     |
| LV-SI    | 1   | —  | 1     |
| LIV-SI   | —   | 1  | 1     |
| SI       | 1   | —  | 1     |
| Total    | 3   | 1  | 4     |

c) Disk herniation between LIII and LIV There was 1 case which showed electromyographic affection of LIV-LV and of all 3 roots clinically

## d) Protrusion between LV and SI

TABLE 27

| Clinical | EMG |        | Total |
|----------|-----|--------|-------|
|          | SI  | Normal |       |
| SI       | 1   | 1      | 2     |
| SI-LV    | 1   | —      | 1     |
| Total    | 2   | 1      | 3     |

e) Protrusion between LIV and LV 1 case with distinct affection of LV clinically as well as electromyographically

f) Protrusion between LIII and LIV 1 case with distinct electromyographic affection of LIV but clinically with affection of all 3 roots

## g) Adhesions between LV and SI

TABLE 28

| Clinical | EMG |       | Total |
|----------|-----|-------|-------|
|          | SI  | SI-LV |       |
| SI-LV    | 1   | 1     | 2     |
| Normal   | 1   | —     | 1     |
| Total    | 2   | 1     | 3     |

## h) Adhesions between L IV and L V

TABLE 29

| Clinical  | EMG |            | Total |
|-----------|-----|------------|-------|
|           | L V | L V → L IV |       |
| L V       | 1   | —          | 1     |
| L V + S I | —   | 1          | 1     |
| Total     | 1   | 1          | 2     |

## i) Adhesions between L V and S I → L IV and L V

TABLE 30

| Clinical  | EMG |           | Total |
|-----------|-----|-----------|-------|
|           | L V | L V + S I |       |
| L V       | 1   | —         | 1     |
| L V + S I | —   | 1         | 1     |
| Total     | 1   | 1         | 2     |

j) Negative operative findings: 1 case which gave a normal electromyogram showed clinical sign of S I affection

Some misleading diagnostic findings compared with the findings at operation occurred in all 3 diagnostic methods. They are discussed below

## I GROUP I DISK CHANGES FOUND AT OPERATION

1) *Misleading clinical neurological findings*

a) Disk herniation between L V and S I: In 9 of these 60 cases of disk herniation weakness of the great toe was the only clinical finding.

In 4 of them there was a lateral disk herniation which affected the L V root (so called lateral L V syndrome described among others by BRADFORD & SPURLING 1939 CRAIG & WALSH 1941 NORLIN 1944 HIRBLIN 1948). In all of these 4 cases electromyography also showed an L V affection (1 of the cases in combination with affection of S I). The myelogram was negative in 2 and showed a lesion between L V and S I in 1 and distally between L IV and L V in 1.

In the other 5 cases there was the usual type of lumbosacral disk herniation with affection of a root regarded as SI at operation. In all of these cases the vertebrae were counted and the number was found to be normal in all. Electromyography showed involvement of SI in 3 cases and normal finding in 1. In these 4 cases the weakness of the great toe may be explained by pain. In the 5th case the electromyogram like the clinical findings suggested affection of LV while the myelogram was of normal appearance. The findings in this case may possibly be explained by an innervation anomaly.

Check examination of the myelogram showed a thickened SI root and filling defect of the SI root sheath.

Finally in 1 case clinical examination showed weakness of the patellar reflex and of the great toe while EMG showed affection of SI root and myelography indicated a lesion between LIV and LV. At operation a spontaneously perforated disk hernia was seen lumbo sacrally and compressing an irritated and a thickened SI root. The disk between LIV and LV was empty and the herniation was certainly referable to this level. Four months had elapsed between myelography and the operation.

c) Disk herniation between LIV and LV. In 9 of the 74 cases of disk herniation between LIV and LV the clinical neurological finding was a changed Achilles tendon reflex (but no weakness of the great toe).

In 4 of these cases the disk herniation was very medial and could therefore satisfactorily explain the affection of the SI root. The only electromyographic finding was also affection of SI in 3 of these cases while the electromyogram was of normal appearance in the 4th. In all 4 cases the myelogram showed a medial herniation between LIV and LV.

Also in the 5th case operation revealed a very medial disk herniation with compression of the SI root. Clinical examination of this case had however shown in addition to affection of SI also involvement of LIV. Electromyography suggested involvement of the SI myotome and myelography showed a slight lumbosacral bulge.

In 1 case electromyography also showed SI denervation and



myelography indicated a lesion between L IV and L V. That patient had, however, 13 thoracic vertebrae.

In 1 case electromyography showed denervation fibrillation in L V as well as the S I myotome. Myelography indicated a lesion between L IV and L V and operation revealed a relatively large spontaneously perforated disk which may have compressed any of the roots or both.

In the 2 remaining cases disk herniation was found at the usual site between L IV and L V. Myelography also indicated a lesion at this level. Electromyography like the clinical examination suggested affection of S I only in one of these cases while in the other case the electromyogram was of normal appearance. The vertebrae were counted and found to be of normal number. In these cases then no other explanation than innervation anomaly can be offered.

c) Protrusions between L V and S I. In 2 of these cases the only clinical neurological finding was weakness of the great toe while electromyography showed denervation in the S I myotome. This might be explained by the following assumption. Weakness of the great toe may have been due to pain, the compression of the S I root produced changes in the sensitive electromyogram but not any clinical signs.

f) Protrusions between L IV and L V. In 1 case with weakness of the Achilles tendon reflex as the only clinical finding, electromyography showed affection of S I. Operation revealed a protrusion with a very medial compression. The myelogram was negative.

## 2) *Misleading myelographic findings*

a) Disk herniation between L V and S I. In 9 cases myelography suggested herniation between L IV and L V while operation revealed lumbosacral herniation.

In 1 of the cases 4 months had elapsed between myelography and operation, the disk had perforated spontaneously into the lumbosacral space, the disk between L IV and L V was empty and the herniation had probably originated from this space.

In 1 of the cases the lumbosacral disk had herniated far out

laterally and affected the LV root and produced a myelographic change of the LV root and its sheath may be even up in the space between LIV and LV

In the other 7 cases no explanation could be found at operation for the misleading findings

c) Disk herniation between LIV and LV In 3 cases myelography showed lumbosacral disk herniation in 2 of them the herniation affected the SI root and thereby *might* have given the changed picture lumbosacrally in the 3rd no explanation was found

In 1 case myelography showed an inward bulge at the level of LIII No explanation was available

As to the protrusions the myelogram was misleading in 2 cases of protrusion between LV and SI and in 2 between LIV and LV Operation revealed no explanation for the changes

### 3) *Misleading electromyographic findings*

a) Disk herniation between LV and SI In 7 cases of lumbosacral disk herniation electromyography showed denervation fibrillation in the LV myotome

In 4 of these cases the herniation was far laterally and pressed against the LV root In these cases clinical examination had also suggested affection of LV (in 1 of the cases in combination with SI affection) In 2 cases the myelogram showed no changes suggestive of a pathological condition in 1 it indicated a lesion between LV and SI and in 1 distally between LIV and LV (so called lateral LV syndrome)

In 1 of the cases the disk herniation was relatively far out laterally and affected the LV root as well as the SI root Clinical examination revealed affection of SI and myelography showed changes between LV and SI

In 1 case No 863/59 operation first revealed a vertical nerve root that was neither swollen nor irritated in the lumbosacral space When the nerve was pricked the patient reported pain in the dorsal part of the calf Another nerve root running obliquely laterally downwards was also observed This nerve root appeared to be swollen and irritated When the nerve was

pricked the patient complained of pain in the great toe. Lateral to this last mentioned nerve root and dislocating it medially was a large (1.5 ml) disk herniation. In this case clinical examination had revealed weakness of the great toe and of the Achilles tendon reflex. The Achilles tendon reflex were however weak on both sides. The myelogram suggested a lumbosacral lesion.

In the 7th case electromyography as well as the clinical neurological examination suggested an affection of IV. Myelography however showed no signs of a pathological condition. The disk herniation was found at the usual place lumbosacrally. Innervation anomaly was the only explanation available. (The number of the vertebrae was normal.)

c) Disk herniation between I IV and LV. In 12 of the patients with disk herniation between I IV and LV electromyography showed denervation in the SI myotome.

In 9 of these cases the herniation was situated medially and pressed against the intradural SI root. Clinical examination showed affection of IV in 1 of the cases, affection of SI in 6 including 2 with combined neurological signs. In 2 cases neurological examination was normal. A medial disk herniation between I IV and LV affecting the SI root has been described by SHIM & WOODS & WIRBEL (1950), CRUI, PUDENZ & SHIMDEN (1957) and KNUTSSON (1958).

In 1 case clinical examination also showed SI affection and myelography indicated a lesion between I IV and LV. That patient had however 13 thoracic vertebrae.

In 1 case the disk herniation had perforated spontaneously and according to the operator it might very well have affected the SI root. Clinical examination had revealed weakness of the great toe.

In the 12th case clinical examination suggested affection of both IV and SI. Operation revealed changes only of the IV root. No explanation was found for the SI denervation and the clinical affection of SI.

d) Protrusions between I IV and LV. In 3 of these cases electromyography showed denervation of the SI myotome as the

only finding. In 2 of these cases operation revealed a definite affection of the SI root by medial pressure of the protrusion. The clinical examination in 1 case was normal and the myelogram indicated changes between LIV and LV. In the 2nd case clinical examination suggested affection of SI while the myelogram showed no lesions.

In the 3rd case clinical examination and myelography showed involvement of LV. The operation revealed a protrusion between LIV and LV with affection of the LV root but lumbosacrally the interspace was narrow and thereby the SI root may have been affected.

## II GROUP 1 NO DISK LESIONS FOUND AT OPERATION

According to Table 14 there were among the "negative" findings 7 cases in which clinical neurological examination was misleading and 3 cases in which myelography and 6 cases in which electromyography had given misleading results.

On analysis of these cases it was found that 1 patient had weakness of the great toe and Achilles tendon reflex. Electromyography had shown LV denervation and myelography had suggested a lesion between LIV and LV but no disk lesions were found at operation. At re-examination 1 year after operation the patient felt better and the weakness of the great toe and denervation of LV had disappeared. There may therefore have been some pressure which was eliminated by the decompressive hemilaminectomy.

In 1 of the cases in which exploration revealed no disk changes electromyographic examination had shown denervation of the SI myotome while the clinical neurological examination and the myelogram both were normal. At the postoperative examination 1 year later the patient was completely recovered and no longer showed any electromyographic changes. Some pressure which could not be demonstrated at operation may therefore have been relieved by the operation.

In 3 other cases in which operation revealed no changes clinical examination and electromyography had shown similar

misleading changes. In 2 of these cases the SI was found affected by both methods. The myelogram was normal in both cases. At re examination 1 year after operation 1 of the patients had not improved and had persistent electromyographic changes, the other patient had improved and the electromyogram was now of normal appearance. In the 3rd case clinical examination as well as electromyography showed affection of both IV and SI while the myelogram showed a thickened IV root. At operation the 2 lowermost spaces were exposed but with negative findings. At re examination the patient showed no improvement.

In 2 cases in which exploration revealed no changes the patients had shown misleading clinical neurological findings but normal electromyograms. In one of these 2 cases clinical examination had indicated affection of the IIV and IV roots and myelography had shown a thickened IV root (and was thus also misleading) in the other clinical examination had shown an affection of IIV but in that patient myelography and electromyography had shown nothing abnormal. These 2 patients showed no improvement at re examination.

In 1 case in which operation had revealed lumbosacral osteophytes pressing a nerve root which was regarded as the SI root clinical neurological examination and electromyography had shown affection of IV while the myelogram was normal. The number of vertebrae was counted and found to be normal. At re examination the patient was better and the weakness of the great toe and the denervation of IV had disappeared.

Myelography was misleading in 1 case in which operation revealed varices lumbosacrally and swollen SI root. Clinical examination and electromyography had also shown SI affection. The myelogram on the other hand indicated a lesion between IIV and IV where operation revealed no changes. At re examination the patient was better and the electromyographic changes had disappeared.

## Group 2

The misleading clinical findings noted in 4 cases may all be due to persistent changes from earlier operation and will therefore not be considered

The patient that showed misleading electromyographic findings had a medially disk herniation between L1<sub>2</sub> and L1 with pressure of the intradural part of the S1 root

As to atrophy and denervation of musculus ext dig brev such changes were found (see chapt III) in 11 of 74 cases of disk herniation between L1<sub>2</sub> and L1 or in 14.9 % while KUGELBERG & PETERSEN (1950) gave such a high frequency as 85 %

In patients with disk herniation between L1 and S1 KUGELBERG & PETERSEN found the same change in only 8 % In the present investigation on the other hand corresponding findings were made in 9 of 60 cases i.e. 15 % This figure appears high therefore these 9 cases are discussed further below

In 4 of the 9 cases there was a lateral disk herniation (so called lateral L1 syndrome) in 1 the herniation was situated relatively laterally and affected the L1 root as well as the S1 root Clinical examination has shown weakness of the Achilles tendon reflex and electromyography had shown L1 denervation In the 6th case the electromyogram and clinical neurological examination also suggested affection of L1

In 2 cases clinical examination showed not only atrophy and denervation of musculus ext dig brev but also weakness of the great toe and of the Achilles tendon reflex

In the 9th case electromyography and clinical neurological examination showed affection of S1

## Chapter V

# THE REVIEW

In order to study the effect of operation and to ascertain whether the electromyographic and clinical neurological changes persisted after the operation the patients were examined clinically and electromyographically 1 year after the operation.

Of the 206 operations 202 were reviewed. Of the 4 who were not reviewed 2 had died in the meantime from some intercurrent disease. The other 2 refused to cooperate.

### I Results

All of the 60 patients with lumbosacral disk herniation co-operated and 33 (55 %) were found to have made a complete recovery. 17 (28.3 %) had symptoms in the form of weakness of the back or back pain but no longer any leg pain. 9 of these 17 wore a corset. 9 (15 %) had persistent sciatic pain but felt better than before the operation. Finally 1 (1.7 %) showed no improvement.

As to the patient in whom operation revealed herniation of the disk between IV and V he had made a complete recovery by the time of the review.

Of the 74 patients who had been operated upon for disk herniation between IV and V 71 (1 died 2 refused to co-operate) were reviewed. 40 (56.3 %) had made a complete recovery while 16 (22.7 %) complained of weakness and pain of the back (9 wore a corset). 11 (15.7 %) felt better but they still had sciatic pain while 4 (5.6 %) showed no improvement at all.

The two patients operated upon for disk herniation between

LIII and LIV were found to have made a complete recovery by the time of the review

As to the 11 patients operated upon for a lumbosacral protrusion 10 were examined (1 had died in the meantime). Of these 10 6 (60 %) had made a complete recovery while 2 (20 %) complained of back pain (1 of them wore a corset) and 2 (20 %) had persistent sciatic pain but felt better than before the operation

The 14 patients who had had a protrusion between LIV and LV all were examined. A complete recovery had been made by 7 (50 %) while 3 (37 %) complained of back pain and 2 of them wore a corset. 1 (7.1 %) felt better but still had sciatic pain and only 1 (7.1 %) reported no improvement at all

All of the 20 in whom operation had failed to reveal any disk changes were reviewed. In the 7 cases in which operation showed osteophytes explaining the root compression syndrome 2 (28.6 %) had made a complete recovery while 3 (42.9 %) felt better but still had sciatic pain and 2 (28.6 %) were unimproved. Of the 4 patients in whom the only operative finding was varices 1 (25 %) had made a complete recovery. 2 (50 %) still had leg pain but otherwise felt better and 1 (25 %) was unimproved. In the case in which operation showed not only varices but also a narrow intervertebral space the patient had no sciatic pain at the review but had back pain and he wore a corset. Of the 8 cases in which operation revealed no signs of a pathological condition at all 1 (12.5 %) had made a complete recovery. 1 (12.5 %) no longer had sciatic pain but persistent back pain (used corset). 3 (37.5 %) felt better but still had sciatic pain and 3 (37.5 %) were unimproved

Of the 24 patients who had been operated upon previously 13 (54.2 %) had made a complete recovery. 2 (8.3 %) no longer had sciatic pain but complained of back weakness (both wore a corset). 7 (29.2 %) were improved but still had sciatic pain and 2 (8.3 %) showed no improvement at all

The results may thus be summarized as follows



*Group 1*

|                                   |                 |          |          |
|-----------------------------------|-----------------|----------|----------|
| Complete recovery                 | 93/178 — 52.2 % | } 75.8 % | } 93.2 % |
| Recovery but persistent back pain | 47/178 — 23.6 % |          |          |
| Improved                          | 31/178 — 17.4 % |          |          |
| Unimproved                        | 12/178 — 6.7 %  |          |          |

*Entire material*

|                                   |                  |          |          |
|-----------------------------------|------------------|----------|----------|
| Complete recovery                 | 106/202 — 52.5 % | } 74.3 % | } 93.1 % |
| Recovery but persistent back pain | 44/202 — 21.8 %  |          |          |
| Improved                          | 38/202 — 18.8 %  |          |          |
| Unimproved                        | 14/202 — 6.9 %   |          |          |

For comparison the literature was studied for the results of operation in previous materials. The analysis showed as follows:

In 1937 BARR reported on 58 cases. 54 (93.1 %) made a complete recovery or improved while 3 (5.2 %) were unimproved and 1 had died from post operative uræmia. The same year LOVE & CAMP reported complete recovery in 66 % improvement in 30 % and no improvement in 4 %. These reports may be regarded as the immediate results of operation. In 1939 CRAIG described a series which he had followed up for 1½—2 years and in which 67 % of the patients were completely recovered while 29 % still had pain of varying degree. Half of these patients were unable to work. In only 5 % were the results really unsatisfactory.

BARR & MIXTER (1941) reviewed 94 of 135 patients operated upon for disk herniation. The interval between operation and the review was at least 1 year (average 1.5 years). They gave the following results: complete recovery 77 % persistent mild symptoms 18 % moderate pain 2 % severe pain 3 %. The same year FRIBERG reported on a review of 24 of 44 patients who were examined 12—27 months after the operation and he reported the results to be satisfactory in 84 %.

The following year MATTHEWS (1942) described a postoperative examination of 54 of 100 patients operated upon for disk herniation. The interval between the operation and the review was 1 year to 6½ years. The results were found to be satisfactory in 70.4 %.

In NORLÉN's (1944) series of 99 patients 62 were reviewed 1—4 years after the operation. Of these 52 (83.9 %) were free from sciatic pain while 10 (16.1 %) still had pain of varying degree.

BRADFORD & SPURLING (1945) reported satisfactory results (good and improved) in 90 %. SELLING & SJÖQVIST (1946) in 92.7 %. FRIBERG & HIRSCH (1946) in 83.8 %. LOVE (1947) in 90.1 %. BARR (1947) in 90 % and LEVNHARD (1947) in 83 %.

In 1948 WARIS reported a review of 347 patients who had been operated upon 1—3 years previously and gave the following results: good 41 %, improved 50 %, unchanged 6 %, worse 1 % and re-operated 2 %. Back pain persisted in 67 %.

In 1950 O'CONNEL published a review of 443 cases who had been operated upon 1½—6 years previously with the following results: recovered 60.7 %, good improvement 31.6 %, improvement 4.7 %, no improvement 0.7 %, recurrence 2.3 %.

Finally in 1952 AITKEN gave a report of a review of 200 patients including 165 in which operation revealed herniation and 35 no disk changes and with the following results:

|           | Disk hernias | No disk changes |
|-----------|--------------|-----------------|
| Excellent | 75 %         | 14 %            |
| Good      | 70 %         | 12 %            |
| Fair      | 21 %         | 26 %            |
| Poor      | 27 %         | 30 %            |
| Bad       | 6 %          | 17 %            |

and ROSS & JELSVIA also in 1952 described 365 patients who had been operated upon 2 years previously. They gave the following results: good 82 %, fair 15.2 %, poor 2.4 %, mortality 0.26 %.

In most of the series then the results were described as satisfactory in more than 90 % as was found in the present material.

## II Clinical findings

The literature contains but scanty data on the persistence or disappearance of neurological signs and symptoms. ALAJOUAINE & PETIT DUTAILLIS discussed this point as early as 1930 and claimed that one should not expect too much of the possi-

bilities of regeneration. The same year (1930) however TOWNE & REICHERT described 2 cases of marked hypertrophy of the ligamentum flavum. 1 of these 2 patients had paresis of the legs, weakened patellar reflex, loss of Achilles tendon reflex and marked impairment of sensibility. 16 months after operation the reflexes and sensibility were normal. In 1937 HOWARD BROWN presented 7 cases with hypertrophy of the ligamentum flavum with the same neurological signs which disappeared after the operation and in 1939 SPURLING, MAYFIELD & ROGERS presented 6 patients in whom the neurological symptoms disappeared before they left hospital. On the basis of experience with a series from the Mayo Clinic ADSON (1940) writes that the injury to the nerve root in some cases was so severe that slight symptoms such as anaesthesia, weakened reflexes and even paresis might persist indefinitely.

MAYNARD (1942) found that slight neurological changes (hyperaesthesia, slight muscular paresis or weakness of reflexes) usually disappeared. But marked impairment of sensibility, paralysis of muscle groups or loss of reflexes often persisted for years.

In 1941 FRIBERG found on review of 24 patients that in 3 of them in whom the patellar reflex was weak before operation this was normal at the review. 2 of these 3 patients showed a weakened Achilles tendon reflex before operation but in those patients the reflex had also become normal by the time of review. In a 4th patient the Achilles tendon reflex was also normal at the review instead of weakened before operation. Thus FRIBERG reported recovery of a weakened reflex in 4 cases.

SPURLING & SJÖQVIST (1946) reported recovery of the Achilles tendon reflex after complete loss in 11 cases. In their series paresis of the extensors of the great toe usually disappeared within a few weeks but in 8 cases paresis persisted for more than a year and in 3 cases for more than 3 years.

In 1946 FRIBERG & HIRSCH reviewed 14 patients who had been operated upon at least 5 years previously. They never found any recovery of the Achilles tendon reflex but regression of the impairment of sensibility in 14 of 33 (42.4 %).

RÖVIG (1949) wrote It is emphasized that neurological changes such as disturbances of the motor and sensory systems and also change in reflexes due to root lesion may persist for years after the compression has been removed

This problem received considerable attention by WARIS (1948) He found that of 56 patients in whom the Achilles tendon reflex had disappeared completely it returned to normal strength in 12 (21.4 %) In 3 (8.9 %) a weak Achilles tendon reflex was demonstrable while in 39 (69.6 %) the loss of the reflex was permanent

Before the operation the Achilles tendon reflex was weak in 18 in 2 (11.1 %) of them the reflex could not be elicited at all at the review 8 (44.4 %) still had a weak reflex and in 8 (44.4 %) it was normal

In 2 cases he also found that complete paresis of the dorsal extensors of the great toe had disappeared by the re examination 15 patients with weakness of the extensors of the great toe were followed up and in 10 (66.6 %) the extensors recovered normal strength and in 5 (33.3 %) slight weakness persisted

As to impairment of sensibility WARIS states that of 137 patients who had had symptom before operation the symptom persisted unchanged in 53 (38.7 %)

At the review WARIS observed no atrophy except in those cases with definitive muscle weakness He writes This may partly support the assumption that the pain in these cases had caused an atrophy of disuse which fairly soon disappears after the pain is relieved and that only the atrophy due to muscle paralysis or weakness has been of a permanent nature On the other hand this disappearance of atrophy is likewise an objective sign of real recovery sciatic pain does not limit the use of extremities and cause atrophy any more

O CONNEL (1950) found persistent reflex changes in 62 % persistent muscular weakness in 66 % and persistent impairment of sensibility in 30 % and atrophy in 8.4 % at review

The neurological symptoms and signs observed 1 year after the operation in the present material are summarized below

1) Achilles tendon reflex

Of 40 patients with loss of the Achilles tendon reflex before operation the reflex was still absent at the review in 34 (85 %) while in 5 (12.5 %) it had returned but was still weak. Only in 1 patient (2.5 %) had the reflex returned and recovered normal strength.

Of 69 patients with weakness of the Achilles tendon reflex before operation the reflex was still weak at the review in 40 (58 %) in 11 (15.9 %) it could not be elicited and in 18 (26.1 %) it had returned to normal.

Of the 4 patients who were not reviewed the reflex was normal in 2 and weak in the other 2 before operation.

#### b) Patellar reflex

In 4 patients in whom the patellar reflex was absent before operation it was also absent at the review.

In 29 the patellar reflex was weak before the operation and in 28 it was still weak after the operation while in 1 it was normal.

Of the 4 patients who were not reviewed the patellar reflex could not be elicited in 1 before operation.

#### c) Paralysis or weakness of the extensors of the great toe

13 of the patients had paralysis of the extensors of the great toe before operation. Of these 6 still had paralysis at the review 4 of them showed weakness of the great toe and in 3 the strength of the toe was normal.

Of the 110 patients with weakness of the gross strength of the extensors of the great toe 84 (76.4 %) showed normal strength of the toe at the re-examination while it was still reduced in 26 (23.6 %).

Of the 4 patients who were not reviewed 3 had had weakness of the extensors of the great toe.

#### d) Impairment of sensibility

Loss of sensibility was noted at the review in 41 (20.3 %) cases against 61 (29.6 %) before the operation. Of the 4 patients who were not reviewed impairment of sensibility had been noted in 1.

#### e) Muscular atrophy

Atrophy of the lower leg and/or the musculature of the thigh

had been noted before operation in 82 (39.8 %) patients against 62 (30.7 %) after the operation. Of the 4 patients not reviewed 2 had had atrophy at the time of the examination before operation.

It must thus be concluded that with the exception of weakness of the dorsal extensors of the great toe the neurological signs to the greatest part persisted unchanged 1 year after the operation.

### III Electromyographic findings

All the patients in the present material with the exception of the 4 who were not reviewed were carefully examined by electromyography 1 year after the operation.

The following findings were made

#### *Group 1*

a) Disk herniation between L<sub>1</sub> and S<sub>1</sub>. Of the 60 patients with lumbosacral disk herniation preoperative examination showed a positive electromyographic finding in 50 (91.7 %). At the review the same electromyographic findings were made in 14 (23.3 %) and in 1 denervation potentials were found only in the medial head of the gastrocnemius.

b) Disk herniation between L<sub>1</sub> and L<sub>VI</sub>. The electromyographic findings were normal at the review but had suggested a pathological condition before the operation.

c) Disk herniation between L<sub>IV</sub> and L<sub>V</sub>. At preoperative examination electromyography suggested changes in 68 (91.8 %) while at the review such changes were only found in 9 (12.7 %) (3 patients not reviewed). In 3 other cases in which the preoperative examination showed denervation of L<sub>V</sub> electromyography at the time of the review showed denervation only of the extensor hallucis in 2 cases and of the extensor hallucis and peroneus longus in the 3rd case. In 1 case denervation potentials were seen only in the erector trunci at the review.

d) The 2 patients with disk herniation between L<sub>III</sub> and

I IV at the preoperative examination showed I IV denervation but no such changes at the review

e) Protrusion between I V and SI Of these 11 patients 10 were re examined and they showed a positive electromyogram in 3 (30 %) while the preoperative examination showed electromyographic abnormalities in 9 (81.8 %)

f) As to protrusions between I IV and I V all 14 showed electromyographic abnormalities before operation as against only 3 at the review (21.4 %)

g) The 20 cases in which operation revealed no disk changes showed electromyographic abnormalities in 14 (70 %) as against 6 (30 %) after operation

### *Group 2*

In this group 22 of the 24 cases showed electromyographic abnormalities (91.7 %) before operation as against only 7 (29.2 %) at the review

At the preoperative examination then 18 (89.8 %) showed electromyographic changes against only 4 (20.8 %) after operation and changes in single muscles in 5 (25 %)

Concerning the electromyographic findings they thus tended to disappear in a great percentage after the operation

A fairly thorough search of the literature failed to reveal any publication on post operative electromyographic examinations of patients who had undergone operation for disk herniation

On the other hand post operative electromyographic studies have been carried out on musculus erector trunci by WFD DELLE IENSTIN & PATTIR (1944) and by MACK (1951) whose works however fall beyond the scope of the present investigation

## DISCUSSION AND CONCLUSIONS

It is clear from chapt IV (Table 12) that in those cases in which operation revealed disk lesions clinical neurological examination had shown the correct level of the lesion in 77 % and the corresponding figure for group 2 *i.e.* patients operated upon previously (Table 13) was 79.2 % and Table 14 (the 20 cases in which operation revealed no disk changes) shows that clinical neurological examination had revealed the correct level of the lesion in 55 %. Clinical neurological examination had thus given a correct diagnosis of the level for the entire material in 76.3 %.

As to the myelographic demonstration of the level of the lesion the examination indicated the proper level for the entire material in 76.1 % (group 1 disk changes in 70.2 % group 2 91.7 % and group 1 no disk changes in 65 %). The corresponding figure for electromyography was 77.6 % (group 1 disk changes in 78.3 % group 2 in 91.7 % and group 1 no disk changes in 50 %). These figures do not include the disk herniation between LV and LVI because it cannot be fitted into a clinical neurological and electromyographic scheme. The myelogram in that case was correct.

It is thus obvious that the 3 methods gave a correct diagnosis with largely equal frequency.

In the comparison of the 3 diagnostic methods the reliability of each method was assessed by comparing each group of operative findings with the diagnostic findings. A more important question from a practical point of view is to what extent the various diagnostic findings can be confirmed at operation.

As to the clinical neurological examination group 1 included



95 cases in which the *Achilles tendon reflex* was weak or absent and in those patients operation revealed a lumbosacral disk herniation in 48 (50.3 %) a lumbosacral protrusion in 8 (8.4 %) osteophytes in the lumbosacral space in 2 (2.1 %) and varices in that space in 1 (1.1 %). Of the patients in whom the *Achilles tendon reflex* was weak or absent in group 1 lumbosacral disk lesions thus were found in 58.9 % and an other explanation for the SI affection in 3.2 %. In group 2 the *Achilles tendon reflex* was weak or absent in 18 cases and of these patients operation showed a lumbosacral herniation in 5 (27.8 %) a lumbosacral protrusion in 3 (16.7 %) and lumbosacral adhesions in 3 (16.7 %). In the entire material operation thus revealed an affection of SI in the lumbosacral space in 62.8 % of the patients in whom the *Achilles tendon reflex* was weak or absent.

In many of these patients however the *Achilles tendon reflex* change was combined with weakness of the great toe and/or changed patellar reflex. If only those cases be considered in which the weakness or absence of the *Achilles tendon reflex* was the only clinical neurological finding (44 cases in group 1 and 6 in group 2) lumbosacral disk changes were seen in 68.2 % (30 cases) in group 1 and another cause of the affection of SI in 4.5 % (2 cases) i.e. a total of 72.7 % (group 1) and of 72 % of the entire material.

Only few authors have described such an analysis of a series. STÅHL (1949) however did give such an analysis of patients with changed *Achilles tendon reflex* as the only finding preoperatively and he found lumbosacral lesions in about 80 % of them at operation. KNUTSSON & WIBERG (1958) gave a corresponding figure of 81 % and FRIBERG & HULT (1951) 77 %.

*Weakness of the dorsal extensors of the great toe* was seen in 110 of the patients in group 1 and operation revealed a herniation between LIV and LV in 56 (50.9 %) of them a protrusion between LIV and LV in 10 (9.1 %) osteophytes in the same space in 1 (0.9 %) and varices in 1 (0.9 %). In these patients with weakness of the dorsal extensors of the great toe then disk lesions were found at operation in the space between

LIV and LV in 60 % of group 1 and in 18 % the LV affection could be ascribed to some other cause. In group 2 10 of the patients had weakness of the great toe and operation revealed a disk herniation between LIV and LV in 1 protrusion between LIV and LV in 1 and adhesions in this space in 2 i.e. a total of 4 cases (26.7 %). Of the entire material then 57.6 % of the patients with weakness of the great toe had a LV affection in the space between LIV and LV.

Weakness of the great toe was however often combined with a change of the Achilles tendon reflex and/or of the patellar reflex. In those cases in which weakness of the great toe was demonstrated as the only clinical neurological finding (33 cases in group 1 and 5 in group 2) operation revealed disk changes between LIV and LV in 75.5 % (40 cases) and some other causal factor for the LV affection in the same space in 3.7 % (2 cases) i.e. a total of 79.2 % (group 1) and of 79.3 % of the entire material.

In patients with weakness of the great toe as the only clinical neurological finding STÅHL found disk lesions between LIV and LV in 83 % HÄUTSSON & WIBERG in 63 % and FRIBERG & HULT in 83 %.

Weakness of the patellar reflex was the only clinical neurological finding in 4 (group 1) and in 2 of them operation revealed a herniation between LIII and LIV. In the other 2 cases operation showed no disk changes. In none of the cases in group 2 was a change in the patellar reflex the only neurological abnormality observed.

*Myelography Group 1* In those cases in which myelography (the primary reports) showed lumbosacral disk herniation (46 cases) the finding was confirmed at operation in 36 and a protrusion was found in 4 thus making a total of 40 (87 %) cases and osteophytes were found in 2 (4.3 %) in the same space. The diagnosis was thus correct in 42/46 (91.3 %).

Myelography showed changes between LIV and LV in 93 cases of which operation showed a disk herniation in the same space in 69 cases and a protrusion in 8 cases thus making a total of 77 (82.8 %) with disk changes and in 4 (4.3 %) the

changes could be explained by other factors (osteophytes in 2 cases and varices in 2) The diagnosis was thus correct in 81/93 cases (87.1 %)

In 2 cases the myelogram showed changes between LIII and LIV and in both of them operation showed disk herniation at that level

*Group 2* In 9 cases of group 2 myelography showed lumbosacral lesions in all of which the diagnosis could be confirmed at operation

In 8 cases myelography showed changes between LIV and LV and in 7 of them these changes were verified at operation

Myelographic changes of disk lesions between LIII and LIV were seen in 2 cases in both of which the changes were verified at operation

The positive myelographic finding could thus be confirmed at operation in the following percentages

Lumbosacral in 92.7 %

Between LIV and LV in 87.1 %

Between LIII and LIV in 100 %

The corresponding figures in FRIBERG & HULT's series were

Lumbosacral in 92.2 %

Between LIV and LV in 89.9 %

Between LIII and LIV in 100 %

*Electromyography* showed SI denervation in 90 of the cases in group 1 48 (53.3 %) of them had a lumbosacral disk herniation and 9 (10 %) were found to have protrusion in the same space the total number of disk lesions was 57 (63.3 %) and another explanation for the SI affection in that space was given in 4 (4.4 %) cases (3 with osteophytes and 1 with varices) making a total of 67.7 % (group 1)

13 of the patients in group 2 had a SI denervation and in these cases operation revealed a lumbosacral disk herniation in 6 (46.2 %) a protrusion in that space in 2 (15.4 %) and adhesions there in 4 (30.8 %) In the entire material thus of the patients with SI denervation 70.8 % had a lumbosacral SI affection at operation

changes could be explained by other factors (osteophytes in 2 cases and varices in 2) The diagnosis was thus correct in 81/93 cases (87.1 %)

In 2 cases the myelogram showed changes between LIII and LIV and in both of them operation showed disk herniation at that level

*Group 2* In 9 cases of group 2 myelography showed lumbosacral lesions, in all of which the diagnosis could be confirmed at operation

In 8 cases myelography showed changes between LIV and LV and in 7 of them these changes were verified at operation

Myelographic changes of disk lesions between LIII and LIV were seen in 2 cases in both of which the changes were verified at operation

The positive myelographic finding could thus be confirmed at operation in the following percentages

Lumbosacral in 92.7 %

Between LIV and LV in 87.1 %

Between LIII and LIV in 100 %

The corresponding figures in TRIBERG & HULT's series were Lumbosacral in 92.2 %

Between LIV and LV in 89.9 %

Between LIII and LIV in 100 %

*Electromyography* showed SI denervation in 90 of the cases in group 1 48 (53.3 %) of them had a lumbosacral disk herniation and 9 (10 %) were found to have protrusion in the same space the total number of disk lesions was 57 (63.3 %) and another explanation for the SI affection in that space was given in 4 (4.4 %) cases (3 with osteophytes and 1 with varices) making a total of 67.7 % (group 1)

13 of the patients in group 2 had a SI denervation and in these cases operation revealed a lumbosacral disk herniation in 6 (46.2 %) a protrusion in that space in 2 (15.4 %) and adhesions there in 4 (30.8 %) In the entire material thus of the patients with SI denervation 70.8 % had a lumbosacral SI affection at operation



LIII and LIV The other 2 showed multiple findings and in 1 of them operation revealed herniation between LIII and LIV

| Total<br>Group 1                                       |    | Disk lesions<br>LIV - SI | Other SI<br>affection<br>LIV - SI | Total  |
|--|----|--------------------------|-----------------------------------|--------|
| Achilles tendon reflex<br>change only                  | 44 | 68.2 %                   | 4.5 %                             | 72.7 % |
| SI denervation only                                    | 70 | 68.6 %                   | 5.7 %                             | 74.3 % |
| Myelographic signs of<br>lesions between LIV<br>and SI | 46 | 87 %                     | 4.3 %                             | 91.3 % |

Thus in 12 patients with a distinct change in the Achilles tendon reflex operation showed no affection of the SI root in the lumbosacral space The following findings were made at operation of these patients disk herniation between LIV and LV in 9 (discussed in chapt IV misleading clinical neurological findings disk herniation between LIV and LV) protrusion between LIV and LV in 1 (discussed in chapt IV misleading clinical neurological findings protrusion between LIV and LV) and no disk changes at operation in 2 (discussed in chapt IV negative findings)

18 patients with a distinct SI denervation showed no lumbosacral SI affection at operation Of these 18 patients the operative findings made were as follows 12 disk herniations between LIV and LV (discussed in chapt IV misleading electromyographic findings disk herniation between LIV and LV) 3 protrusions between LIV and LV (discussed in chapt IV misleading electromyographic findings protrusion between LIV and LV) and 3 no disk changes at operation (discussed in chapt IV neg operative findings)

In 4 patients myelographic changes between LIV and SI could not be confirmed at operation 3 of them were found to have herniation between LIV and LV (see chapt IV misleading myelographic findings disk herniation between LIV and LV) and 1 of them a protrusion between LIV and LV (see chapt IV misleading myelographic findings protrusion between LIV and LV)

protrusion between L<sub>4</sub> and S<sub>1</sub>) and in 1 no disk changes (see chapt IV negative findings)

| Total<br>Entire material  | Disk lesions<br>L <sub>4</sub> - L <sub>5</sub> | On r L <sub>4</sub><br>aff. L <sub>4</sub> - L <sub>5</sub> | Total  |
|---|---|---|--------|
| Weakness of the great<br>toe only   | 58  | 74.1 %  | 79.3 % |
| L <sub>4</sub> denervation only   | 75  | 80 %  | 86.7 % |
| Myelographic signs of<br>lesions between L <sub>4</sub><br>and L <sub>5</sub> | 101   | 81.2 %  | 86.1 % |

A question of practical importance is to what extent all three diagnostic methods give single or combined or normal findings respectively in the same cases or whether the three methods differ from one another in this respect. The relationship between single combined and normal findings respectively on clinical neurological and electromyographic examinations is apparent from Tables 19—30 in chapt IV and therefore need not be discussed again here. The ratio between these findings and the myelographic findings is as follows:

*Group 1 a) Disk herniation between L<sub>4</sub> and S<sub>1</sub>* In 5 patients in which electromyography showed no signs of pathological condition the myelogram showed lumbosacral changes in 4 but not in the 5th (in this case however the clinical neurological examination indicated an affection of S<sub>1</sub>)

In 2 cases the clinical neurological findings were normal and in 1 of them the myelogram indicated lumbosacral changes while in the other case it revealed no signs of a pathological condition (in this case however the electromyogram showed S<sub>1</sub> denervation)

Of the 14 cases in which myelography was of normal appearance in patients with lumbosacral disk herniation found at operation electromyography and clinical neurological examination distinctly showed the same lesions in 6 of them. In 3 of the 14 cases electromyography gave clear cut information while the clinical neurological examination gave a combined finding while in 2 cases it was the converse. In 1 case there were combined findings on electromyographic as well as clinical neuro

The group included 3 patients in whom myelography showed no signs of a pathological condition and 1 in which the myelographic findings were suspected but in all of these cases clinical neurological and electromyographic examination gave a firm diagnosis.

*Group 2* Of the positive findings made at operation in group 2 re-operated cases, electromyography was of normal appearance in one case, a protrusion between LV and SI but in this case the myelogram suggested lumbosacral lesion and in 1 case there was a normal clinical neurological finding, namely, in the subgroup adhesions between LV and SI and in that case the myelogram was normal too.

Of the patients (in group 2) in whom operation revealed positive findings, the latter had been missed by myelography in 3, namely in the one referred to above in which the electromyogram showed lesion of SI and thus was the only positive finding by the 3 diagnostic methods. In the same subgroup (adhesions between LV and SI) myelography was normal in another case too in which the electromyogram indicated lesion of SI while the clinical neurological examination suggested affection of both LV and SI. The 3rd case where the myelography was of normal appearance belonged to the subgroup disk herniation between LV and SI and in that case the clinical neurological examination suggested lesion of the LV root while the electromyogram indicated affection of both the LV and the SI myotomes. This case is described here because reoperation had been done partly because of the electromyographic findings. The patient was a 30 year old male operated upon on 6.11.1957, when a large herniation of the disk (2.2 ml) was found between the 4th and 5th lumbar vertebrae. The patient however was not relieved of his radiating pains and he came back to the hospital in January 1958. Neurological examination showed the same situation as before the first operation, namely paresis of the great toe, Lasague's test positive at  $30^{\circ}$ . Myelography was negative. Electromyography showed denervation fibrillation in both the LV and SI myotomes. A new operation revealed a herniation of the disk (0.6 ml) in the lumbosacral space.



and since its removal the patient has been free of pain (case 18a/58)

Summarizing in a few of those cases in which operation revealed changes only one of the examination methods gave a positive finding. Thus *clinical neurological* examination showed lumbosacral disk herniation in one case and lumbosacral protrusion in another in which cases both myelography and electromyography had been of normal appearance.

As to the *myelographic* examination this in 2 cases was the only method which showed disk herniation between LIV and LV.

Finally *electromyographic* examination was the only method that revealed signs of a pathological condition in 1 case of disk herniation between LV and SI (group 1) in 1 case of protrusion between LV and SI (group 2) and in the described reoperated case with a disk herniation between LV and SI found at the reoperation. In none of the cases in which operation revealed disk changes did all examinations fail to detect the lesions.

The 20 cases (in group 1) with no disk changes found at operation are discussed below.

This group included 8 cases in which operation revealed no changes at all.

In 1 of these patients none of the 3 examination methods had revealed any signs of disease but the patient had severe pain and atrophy of the thigh and calf muscles for which reason explorative surgery was decided upon but the results were negative (apart from a possible thickening of the SI root). At re-examination 1 year after the operation the patient reported no improvement but still showed no electromyographic or clinical neurological abnormalities.

In 1 case clinical examination as well as electromyography had shown changes of both the LV and the SI root while the myelogram suggested a thickened LIV root. The 2 lowermost spaces were explored but revealed nothing of interest. At re-examination the patient felt no better and the clinical and electromyographic changes were still demonstrable.

In 1 of the cases both clinical examination and electromyography showed affection of SI and the myelogram suggested lumbosacral lesions. The 2 lowermost disks were explored but no changes were found. At re-examination the patient was unimproved and the clinical neurological and electromyographic changes were still demonstrable.

In 2 other cases in which the patients were likewise unimproved at the review 1 of the patients had clinical neurological symptoms of affection of LIV but neither myelography nor electromyography showed any abnormalities and in that case the 3 lowermost spaces were explored but nothing of interest was found and in 1 patient with clinical affection of the LIV and the LV root myelography showed a thickened LV root but the electromyogram was normal and in that case exploration of the 2 lowermost spaces revealed no changes either.

In the 5 cases, described above it is possible that the surgeons might have missed a possible cause of root compression.

Of the remaining 3 patients in whom surgical exploration revealed no changes 2 were improved and 1 had made a complete recovery by the time of the review. One of the improved patients showed clinical and electromyographic evidence of affection of SI but no myelographic changes and the other patient showed clinical evidence of involvement of the LV and the SI root. Electromyographic signs of LV denervation and myelography showed changes in the space between LIV and LV. In both cases the electromyographic changes as well as the weakness of the great toe in the last case had disappeared by the time of the review. The patient who had made a complete recovery had shown no clinical neurological signs before operation and the myelogram had been normal but electromyography had shown SI denervation. The electromyographic abnormality had disappeared by the time of the review.

In these 3 last mentioned cases there had either been spontaneous restitution or some compression might have been present which could not be demonstrated at operation but which was nevertheless relieved by the decompressive hemilaminectomy.

In 7 cases osteophytes were seen that could explain the root compression

In 1 of these cases the change was only demonstrable by myelography while clinical neurological examination and electrography revealed nothing of interest. The patient had improved by the time of the review.

In 4 cases all 3 examination methods showed root compression. 2 of these patients had completely recovered by the time of the re-examination and by them the electromyographic changes had disappeared while the other 2 were unimproved and still showed electromyographic abnormalities.

In a 6th case myelography showed abnormalities while the compression produced both clinical neurological and electromyographic signs. At the review this patient had improved and then both the clinical neurological sign (weakness of the great toe) and the denervation had regressed.

In the 7th case the clinical neurological examination revealed no abnormalities while myelography and electromyography suggested the same lesions. The patient felt better at the review but the denervation was still demonstrable.

From the observations made in these 7 cases it may be concluded that the osteophytes *may* cause root compression and that their removal *may* be followed by improvement or even by complete recovery.

Of the 20 cases (group 1) in which operation revealed no disk changes there were still 3 in which the operative findings were described as varices in 4 and as varices + narrow space in the 5th. Before operation the last mentioned patient showed L4 denervation and myelography suggested a change in the space between L4 and L5 while the clinical neurological examination showed weakness of the patellar reflex. At the review the patient had made a complete recovery and the L4 denervation had disappeared. Unless this recovery had been spontaneous the L4 root might have been compressed by the very narrow space observed at operation where according to the surgeon the arches almost contacted one another and where large varicose veins were seen.

In 1 of the cases both clinical examination and electromyography showed affection of SI and the myelogram suggested lumbosacral lesions. The 2 lowermost disks were explored but no changes were found. At re-examination the patient was unimproved and the clinical neurological and electromyographic changes were still demonstrable.

In 2 other cases, in which the patients were likewise unimproved at the review, 1 of the patients had clinical neurological symptoms of affection of LIV but neither myelography nor electromyography showed any abnormalities and in that case the 3 lowermost spaces were explored but nothing of interest was found and in 1 patient with clinical affection of the LIV and the LV root myelography showed a thickened LV root but the electromyogram was normal and in that case exploration of the 2 lowermost spaces revealed no changes either.

In the 5 cases described above it is possible that the surgeons might have missed a possible cause of root compression.

Of the remaining 3 patients in whom surgical exploration revealed no changes, 2 were improved and 1 had made a complete recovery by the time of the review. One of the improved patients showed clinical and electromyographic evidence of affection of SI but no myelographic changes and the other patient showed clinical evidence of involvement of the LV and the SI root, electromyographic signs of LV denervation and myelography showed changes in the space between LIV and LV. In both cases the electromyographic changes as well as the weakness of the great toe in the first case had disappeared by the time of the review. The patient who had made a complete recovery had shown no clinical neurological signs before operation and the myelogram had been normal but electromyography had shown SI denervation. The electromyographic abnormality had disappeared by the time of the review.

In these 3 last mentioned cases there had either been spontaneous restitution or some compression might have been present which could not be demonstrated at operation but which was nevertheless relieved by the decompressive hemilaminectomy.

d) Varicosis may be the result of over abundant blood supply e.g. through vascular tumours angiomatosis of the central nervous system and haemangiomatosis vertebrae

In 1 case in group 2 surgical exploration revealed no changes at all. Before operation the Achilles tendon reflex was possibly somewhat weak but neither electromyography nor myelography had shown any signs of a pathological condition. 6 years previously the same space had been explored and neither then had operation revealed any changes. By the time of the review 1 year after the last operation the patient had recovered and the Achilles tendon reflex was normal.

Also among some of those cases in which operation revealed no disk changes only one of the methods had suggested disease, namely the clinical neurological examination in 2 cases in which the operator found only varices and in which one of the patients made a partial recovery and the other complete recovery after explorative surgery and in 2 cases in which operation revealed no changes at all. In one of these cases (group 2) the patient had recovered and in the other (group 1) he showed no improvement at all by the time of the review.

The myelographic examination revealed changes in 1 case in which operation showed osteophytes and in which the patient improved after the operation.

Electromyography showed lesions in 1 case in which exploration revealed no changes at all and in which the patient made a full recovery with disappearance of the electromyographic changes after the operation.

In 1 case none of the diagnostic methods showed any signs of disease and surgical exploration provided no explanation either. At the review that patient was unimproved.

As is apparent from chapt. V the postoperative regression was considerable as far as electromyographic changes are concerned while the clinical neurological findings to a large degree persisted. For practical reasons myelography was not done after the operation. No correlation was found between the postoperative regression of the electromyographic changes and the size of the disk hernia or the results of operation.

The findings described above suggest the following conclusions

I Neurological examination and electromyography cannot always give an exact diagnosis. The reasons for this inexactitude are as follows

a) The IV root can be compressed also by a lateral prolapse in the IV and SI space as shown by the 4 patients in which the disk had herniated far laterally in the lumbosacral space (see chapt IV)

b) The SI root can be compressed by a medial prolapse in the LIV—LV space. The present material included 4 cases of medial disk herniation between LIV and LV with clinical as well as electromyographic changes suggesting only involvement of SI. And this also applies to a medial protrusion between LIV and LV (see chapt IV)

c) One and the same prolapse can compress two roots and thus give combined findings (see analysis in chapt IV). There are for example cases with clinical neurological as well as electromyographic signs of affection of both the IV and the SI root and in which the electromyographic changes had disappeared by the time of the review

d) The lowermost disk is not always the 24th from the atlas. For example 1 patient may have 7 cervical vertebrae, 12 thoracic vertebrae and 6 lumbar vertebrae such as the patient who was classified as having a disk herniation between LV and LVI

e) Individual variations occur in the nervous system. In the present material there was 1 case of lumbosacral disk herniation at the usual place in the spine with clinical and electromyographic findings suggesting only involvement of LV and 1 case of disk herniation between LIV and LV at the usual place in the disk with only clinical and electromyographic signs of involvement of SI (see chapt IV). In both these cases the electromyographic changes had disappeared by the time of the review

II In certain cases 2 or 3 roots may produce clinical neurological signs while the electromyographic examination may show only involvement of 1 root and vice versa. This is illustrated by Tables 19—30 in chapt IV

III Myelography is not exact either because lateral prolapses can compress a root without causing any defect in the contrast column in the dural sac or root pockets. It was pointed out in the section on the myelographic findings that at the level of the disk between L4 and S1 the dural sac often tapers so that large lateral prolapses may be present despite a normal myelogram.

In view of the above considerations it appears that electromyography is valuable in the diagnosis of the lumbar root compression syndrome and on the basis of the analysis performed in the present material its importance may be summarized as follows:

- 1) The electromyogram may show changes in the absence of clinical neurological findings (see Tables 19—30 in chap. IV).
- 2) The electromyogram may show involvement at one level when combined clinical neurological symptoms may suggest changes at two levels (see Tables 19—30 in chap. IV).

These 2 conclusions are illustrated by Tables 19—30 in chap. IV.

- 3) The electromyogram may show abnormalities when the myelogram is normal.

Of the 14 patients in whom myelography showed no changes but in which operation revealed lumbosacral disk herniation the electromyogram showed changes in 13 and of the 4 patients with normal myelograms but with the operative findings protrusion between L4 and S1 3 showed a positive electromyogram. As to the space between L4 and L5 the myelograms were normal in 4, 1 in whom operation revealed disk herniation and 3 in which operation showed protrusion. In all of these 4 cases electromyographic changes were noted. The degree of accord between myelography on one hand and electromyography and clinical neurological examination on the other hand among patients in whom no disk changes could be found at operation were discussed in a previous section. Group 2 included 2 patients in whom operation revealed lesions that had not been demonstrated in the myelogram but in the electromyogram.

4) The electromyographic changes regress considerably after operation and can therefore be used with advantage for demonstration of so called recurrences. This is apparent from the postoperative examination which is accounted for in chapt. V. The clinical neurological changes, with the exception of weakness of the great toe, were still demonstrable in a high degree 1 year after the operation, while the electromyographic changes persisted in only about one fourth of the patients.

The disadvantages of electromyography are as follows:

1) The electromyographic denervation potentials do not appear until 18—21 days after onset of the disease.

2) Every muscle must be examined very carefully, requiring the insertion of the needle a relatively large number of times.

There appears to be general agreement on the value of electromyography for the diagnosis of lumbar nerve root compression syndrome. SHLA & WOODS (1955) write: "Perhaps the commonest and most practical clinical use of the LMG is as an aid in the diagnosis of a compression lesion of a specific nerve root (most frequently by a protruded intervertebral disk)." MARINACCI (1955) writes: "The LMG has proved itself to be of definite significance in determining and localizing nerve root lesions."

The clinical findings, when correlated with the LMG, aids the surgeon in selecting his cases for opaque myelography. And in his book on "Clinical Electromyography" MARINACCI (1955) concludes: "In the diagnosis of nerve root compression syndrome the EMG is slowly but surely replacing subjective uncertainty with objective reality. Nevertheless the LMG is a laboratory procedure and its findings must always be correlated with the history and the clinical picture."

It is clear from the present investigation that electromyography is not superior to the other two methods, but that it is a good supplementary method.



## Chapter VII

# SUMMARY

A series of 206 cases (203 patients) operated upon for lumbar nerve root compression syndrome was studied with respect to the diagnostic value of clinical neurological myelographic and electromyographic examination

The operative findings made in the material were

|  |                  |
|--|------------------|
| <i>Group 1</i> (patients not operated upon previously)                                     | 182              |
| a) Disk herniation between L <sub>5</sub> and S <sub>1</sub>                               | 60               |
| b) Disk herniation between L <sub>5</sub> and L <sub>VI</sub>                              | 1                |
| c) Disk herniation between L <sub>IV</sub> and L <sub>V</sub>                              | 14               |
| d) Disk herniation between L <sub>III</sub> and L <sub>IV</sub>                            | 2                |
| e) Protrusion between L <sub>5</sub> and S <sub>1</sub>                                    | 11               |
| f) Protrusion between L <sub>IV</sub> and L <sub>V</sub>                                   | 14               |
| g) No disk changes   | 20               |
| consisting of 4 subgroups  |                  |
| 1) No pathological findings at operation   | 8                |
| 2) Osteophytes   | 7                |
| 3) Varices   | 4                |
| 4) Very narrow interspace + varices  | 1                |
| <i>Group 2</i> (previously operated cases)   | 24 (23 patients) |
| a) Disk herniation between L <sub>5</sub> and S <sub>1</sub>                               | 6                |
| b) Disk herniation between L <sub>IV</sub> and L <sub>V</sub>                              | 4                |
| c) Disk herniation between L <sub>III</sub> and L <sub>IV</sub>                            | 1                |
| d) Protrusion between L <sub>5</sub> and S <sub>1</sub>                                    | 3                |
| e) Protrusion between L <sub>IV</sub> and L <sub>V</sub>                                   | 1                |
| f) Protrusion between L <sub>III</sub> and L <sub>IV</sub>                                 | 1                |
| g) Adhesions between L <sub>5</sub> and S <sub>1</sub>                                     | 3                |
| h) Adhesions between L <sub>IV</sub> and L <sub>V</sub>                                    | 2                |
| i) Adhesions between L <sub>5</sub> and S <sub>1</sub> +L <sub>IV</sub> and L <sub>V</sub> | 2                |
| j) No pathological findings at operation   | 1                |

An account is then given of projected pain in the different groups. Projected pain into the heel was noted in 20 % of the

equivocal positive diagnostic findings and the operative findings and it was found that in group 1 operation revealed lumbosacral changes in 72.7 % of all patients (44) with an unequivocal Achilles tendon reflex change or in 72 % of the entire material, operation revealed lumbosacral changes in 74.3 % of all patients (70) in group 1 with definite SI denervation or in 75.9 % of the entire material and that if the myelogram showed lumbosacral changes these changes were confirmed at operation at the same level in group 1 in 91.3 % or in 92.7 % of the entire material and that operation revealed changes between LIV and LV in group 1 in 79.2 % of all patients with paresis of the great toe or in 79.3 % of the entire material operation revealed changes between LIV and LV in 85.5 % of all patients with unequivocal LV denervation in group 1 or in 86.7 % of the entire material Myelographic changes between LIV and LV were confirmed at operation in the same space in 87.1 % of group 1 and in 87.1 % of the entire material

In addition those cases in which operation revealed no disk changes are analysed Finally it is given an account of the possibilities of the various diagnostic methods

**Acknowledgements** I wish to express my sincere thanks to Professor Gunnar Wiberg M.D. for generous help, advice and encouragement throughout the investigation

Valuable help in various ways and kind co-operation was offered by Professor Gunnar Wohlfart M.D. I thank him heartily

Thanks go to Laborator Olof Norman and Doctor Sten Cronqvist for invaluable help with the roentgenexaminations

I feel deeply indebted to Docent Folke Ståhl for constructive criticism and stimulating discussions

The investigation was supported by grants from the Medical Faculty University of Lund

# REFERENCES

- ABBOT W D Compression of cauda equina by the ligament flavum  
JAMA 1936 106 2129—2130
- ACCORNEO A Observation sur le réflexe achilléen dans la sciatique Rev  
neuro 1911 27 (11) 805—807
- ADRIAN E D and BRONK D W The discharge of impulses in motor nerve  
fibres II The frequency of discharge in reflex and voluntary contrac  
tions J Physiol 1929 67 119—151
- ADSON A W Bandscheibenerreissung mit Prolaps des Nucleus pulposus  
in den Wirbelkanal als Ursache rezidivierender Ischias Der Chirurg  
1940 12 501
- ADSON A W and OTT W O Results of the removal of tumors of the  
spinal cord Arch. Neurol & Psychiat 1922 8 570—538
- AITKEN A Rupture of the intervertebral disc in industry further observa  
tions on the end results Am J Surg 1939 84 261—267
- AITKEN A P and BRADFORD C H End result of ruptured intervertebral  
discs in industry Am J Surg 1947 3 365—380
- ALAQUANINE T et PETIT DUTAILLIS D Compression de la queue de che  
val par une tumeur d'un disque intervertébral ablation suivie de  
guérison. Bull et mém Soc nat de chir 1929 55 937—945  
— La nodule fibrocartilagineux de la face postérieure des disques inter  
vertébraux Presse méd 1930 38, 937—945
- ANDERSEN T och CHROM S A Luftmyelografi hos ischiasspäsenter Norl  
Med 1949 14 1619—1626
- ANDRAE R Über Knorpelknötchen an hinteren Ende der Wirbelbandschei  
ben im Bereich des Spinalkanals Beitr path Anat 1899 82 464—474
- ARNELL S Weitere Erfahrungen über Myelographie mit Abrodil Acta  
Radiol 1944 25 408—413  
— Myelography with water soluble contrast Acta Radiol 1948 suppl 75
- ARNELL S and LIDSTRÖM F Myelography with Skiodan (Abrodil) Acta  
Radiol 1931 17 187—289
- AYERS C E Lumbo sacral backache New England J Med 1929 200  
592—608
- BABINSKI J 1876 quoted by Norlen
- BADGLEY C E Clinical and roentgenological study of low back pain with  
sciatic radiation clinical aspects Am J Roentgenol 1937 37 454—  
460

equivocal positive diagnostic findings and the operative findings and it was found that in group 1 operation revealed lumbosacral changes in 72.7 % of all patients (44) with an unequivocal Achilles tendon reflex change or in 72 % of the entire material operation revealed lumbosacral changes in 74.3 % of all patients (70) in group 1 with definite SI denervation or in 75.9 % of the entire material and that if the myelogram showed lumbosacral changes these changes were confirmed at operation at the same level in group 1 in 91.3 % or in 92.7 % of the entire material and that operation revealed changes between LIV and LV in group 1 in 79.2 % of all patients with paresis of the great toe or in 79.3 % of the entire material operation revealed changes between LIV and LV in 85.5 % of all patients with unequivocal LV denervation in group 1 or in 86.7 % of the entire material Myelographic changes between LIV and LV were confirmed at operation in the same space in 87.1 % of group 1 and in 87.1 % of the entire material

In addition those cases in which operation revealed no disk changes are analysed Finally it is given an account of the possibilities of the various diagnostic methods

*Acknowledgements* I wish to express my sincere thanks to Professor Gunnar Wiberg M.D. for generous help, advice and encouragement throughout the investigation

Valuable help in various ways and kind co-operation was offered by Professor Gunnar Wohlfart M.D. I thank him heartily

Thanks go to Laborator Olof Norman and Doctor Sten Cronqvist for invaluable help with the roentgenexaminations

I feel deeply indebted to Docent Folke Stahl for constructive criticism and stimulating discussions

The investigation was supported by grants from the Medical Faculty University of Lund

- BECHTAHL F An introduction to electromyography Kbhvn Guldendal 1957
- BACA P C Chondroma of intervertebral disc JAMA 1950 94 1557—1558
- BENTS A T Surgical aspects of rupture of intervertebral disc Radiol 1941 36 604—616
- BLOOM DEL M S Die lumbale Myelographie zur Diagnose der Diskushernie Fortschr Röntgenstr 1957 87 334—34
- BURT J B Problems in the study of sciatica Brit M J 1934 2 188—89
- BUSCH E Om luftmyelografi ved lumbosacral discsprellaps og ligamentær compression Ugeskr f læger 1940 10 899—907
- BUSCH E und CHRISTENSEN E Die lumbalen Lufposushernien Zentralbl f Neurochir 1936 1 53—58
- CAPLA A Encephalography with subarachnoidal injection of thorotrast Radiol med 1933 20 136—138
- CHAMBERLAIN E W and YOUNG B I The diagnosis of intervertebral disc protrusion by intraspinal injection of air JAMA 1939 113 9072—9074
- CLARK D A Muscle counts of motor units A study in innervation ratios Am J Physiol 1931 96 296—304
- COTUNAR D De ischiade nervosa commentarius Rudolphum Craff r Viennæ 170 (1764)
- CRAIG W Mck The present status of the protruded disc Compt rend III Congres Neurolog Intern Copenhagen 1909 57—54
- CRAIG W Mck and WALSH M N Neuroanatomical and physiological aspects and significance of sciatica J Bone & Joint Surg 1941 23 117—134
- CROLE B L FLDENZ R H and SHILLEN C H Observations on the value of clinical electromyography J Bone & Joint Surg 1957 39 A 492—500
- DANDY W E Ventriculography following injection of air into cerebral ventricles Ann Surg 1918 68 5
- Loose cartilage from intervertebral disc simulating tumour of the spinal cord Arch Surg 1929 19 660—662
- Concealed ruptured intervertebral discs JAMA 1941 11 871—823
- Improved localization and treatment of ruptured intervertebral discs JAMA 1947 190 605—660
- Recent advances in the treatment of ruptured (lumbar) intervertebral discs Ann Surg 1943 118 639—645
- Recent advances in diagnosis and treatment of ruptured intervertebral discs Am J Surg 1947 115 514—520
- DANFORTH M S and WILSON P D The anatomy of the lumbosacral

- region in relation to sciatic pain J Bone & Joint Surg, 1925 7 109—160
- DAUM H I SMITH A B WALKER J W, CHALMAN S B and LIVERSMAN G H Protrusions of the lumbar disc A correlation of the radiographic diagnoses and surgical findings Southern Med J 1933 22 143—1484
- DEJERINE J Les radiculites Rev neurol 1916 29 321—348
- Séméiologie des affections du système nerveux Masson et C<sup>ie</sup> Paris 1926 1219 pp
- DEJERINE J et REGNARD M Sciatique radiculaire avec paralysie dissociée des muscles antéro externes de la jambe droite Intégrité du jambier antérieur Anesthésie dans le territoire de S I Rev neurol 1912 23 288—290
- DENNY BROWN D On the nature of postural reflexes Proc Roy Soc London 1929 61 99—178
- DENNY BROWN D and PENNYBACKER J B Fibrillation and fasciculation in voluntary muscle Brain 1938 61 311—334
- DUBARRY L C L R Contribution à l'étude de la sensibilité cutanée dans la névralgie sciatique P Cassagnol Bordeaux 1902 94 pp
- ECCLES J C and SHERRINGTON C S Numbers and contraction values of individual motor units examined in some muscles of the limb Proc. Roy Soc London 1930 106 376—357
- EKVALL, S Enquête clinique au printemps de 1938 sur les cas de sciatique observés durant les années 1933 et 1934 Acta Med Scand 1939 101 1—33
- ELLSBERG L A Experiences in spinal surgery Surg Gynec & Obst 1913 16 117—132
- Diagnosis and treatment of surgical disease of the spinal cord and its membranes N B Saunders & Co Philadelphia and London 1916
- Tumours of the spinal cord Paul B Hoeber New York 1925
- Extradural ventral chondromas (ecchondroses) their favorite sites spinal cord and root symptoms they produce and their surgical treatment Bull Neurol Inst New York 1931 1 350—388
- Surgical diseases of the spinal cord membranes and nerve roots H K Lewis and Co London 1912 528 pp
- FASSBENDER C W, HILSSER G und STÜSSEL, H G Über die Ergebnisse der Abrodil Myelographie beim lumbalen Bandscheibenvorfall Fortschr Röntgenstr 1938 89 5 574—578
- FELTSTRÖM E Myelografi med abrodil Nord Med 1942 14 1652—1655
- FERNET C De la sciatique et de sa nature Arch gen d méd 1848 1 385—403
- FINCHER L I Neurologic aspects of low back pain and sciatica Ann Surg 1933 109 1028—1033
- FLORES, A Compression médullaire progressive a 6 ans de durée par

- hyperplasie du ligament jaune consecutive a fracture d'une lame vertébrale Laminectomie guérison *Rev neurol* 1923 30 664—667
- FOERSTER O Symptomatologie der Erkrankungen des Rückenmarks und seiner Wurzeln. Im Bunte und Foerster Handbuch der Neurologie 1926 5 1—403
- FORD L T and KEY J A An evaluation of myelography in the diagnosis of intervertebral disc lesions in the low back *J Bone & Joint Surg* 1941 23 417—434
- FORD L T, RAMSEY R H., HOLT E P and KEY J A An analysis of 100 consecutive lumbar myelograms followed by disc operations for relief of low back pain and sciatica *Surgery* 1952 32 961—966
- FORESTIER M H Pathogénie sacro vertébrale de certaines sciaticques Bull gen de therap 1914 167 484—492
- FREIBERG A H Sciatic pain and its relief by operations on muscle and fascia *Arch Surg* 1937 34 337—350
- FREIBERG A H and VINKE T H Sciatica and sacro iliac joint *J Bone & Joint Surg* 1934 16 126—136
- FREIBERG S Low back and sciatic pain caused by intervertebral disc herniation *Acta Chir Scand* 1941 85 suppl. 64, 1—114
- FREIBERG S and HIRSCH C On late results of operative treatment for intervertebral disc prolapses in the lumbar region. *Acta Chir Scand* 1946 93 161—169
- FREIBERG S and HULT L Comparative study of a brodif myelogram and operative findings in low back pain and sciatica *Acta Orthop Scand* 1951 20 303—314
- GALVANI L De viribus electricitatis in motu musculari commentarius Memoirs of the Institute of Sciences Bologna Italy 1 91 7
- GARA, S Über ein bisher unbekanntes pathognomonisches Symptom der Ischias *Wien med Wchnschr* 1907 57 1135—1139
- GHORMLEY R h Low back pain *JAMA* 1933 101 173—177
- GOLDTHWAIT J E The lumbo sacral articulation An explanation of many cases of lumbago sciatica and paraplegia *Boston Med and Surg J* 1911 164 565—572
- GOLDTHWAIT J E and OSGOOD R B A consideration of the pelvic articulations from an anatomical, pathological and clinical standpoint *Boston Med and Surg J* 1905 152 593—601 634—638
- GOLSETH, J G Diagnostic contributions of the electromyogram *Calif Med* 1950 3 355—357
- GOLSETH J G and FIZZELL J A Electromyographic studies on cats after section and suture of the sciatic nerve *Am J Physiol* 1947 150 558—567
- GOWERS W R A manual of diseases of the nervous system London J & A Churchill 1892 vol I 101—108

- GUINON G and LARMENTIER E Sur une complication peu connue de la sciastique Arch d neurol 1890 20 145—186
- VON HAGEN K O The clinical application of electromyography to lower motor neuron and muscular disorders Bull Los Angeles Neur Soc 1955 20 131—138
- HAGGART C L Sciatic pain of unknown origin effective method of treatment J Bone & Joint Surg 1938 20 851—859
- HANRAETZ P R M J The degenerative back Elsevier Publ Co Amster dam 1959
- HELLEDAY U On myelitis chronica Nord Med Ark 1896 8 1—17
- HELWEG J Ischias en klinisk studie koebenhavn Henrik Koppel 19 0 272 pp
- HERRLIN L The lateral fifth lumbar root syndrome in sciatica Opusc Med 1958 3 85—91
- HELMAN G Muskelreumatism Sv Lak tidn 1920 17 185—194  
— Än en gång sjukdomar i ryggraden såsom orsak till lumbago och ischias Sv Lak tidn 1920 17 457—460
- HIBBS R A and SWIFT W E Developmental abnormalities at the lumbosacral juncture causing pain and disability Surg Gynec. & Obst 1979 48 604—612
- HIRSCH C Intervertebral foraminotomy Acta Chir Scand 1946 94 55—60  
— The clinical evaluation of sciatica Acta Orthop Scand 1958 27 210—218
- HOEFER I F A and GUTTMAN S A Electromyography as a method for determination of level of lesions in the spinal cord Arch Neurol & Psychiat 1944 51 415—422
- HOEFER P F A and PUTNAM T J Action potentials of muscles in normal subjects Arch Neurol & Psychiat 1939 42 201—218
- HILBERT VALLEROUX 1870 quoted by Dubarry
- HUDDLESTON O L and GOLSETH J G Electromyographic studies of paralyzed and paralytic muscles in anterior poliomyelitis Arch Phys Med 1948 29 97—98
- INGVAB S On the nature of myalgia Acta Rheum 1936 8 30—31
- JACOBI W, LÜHR, W and WESTMAN O Über die Darstellung des zentralen und peripheren Nervensystems im Röntgenbild Leipzig J A Barth 1934
- JASPER H H Report to Associate Committee on Army Medical Research National Research Council C 6239 Canada Montreal Neurological Institute McGill University 1945
- JASPER, H H and JOHNSTON R H A portable clinical electromyograph Report to Associate Committee on Army Medical Research National Research Council Canada Montreal Neurological Institute McGill University 1945



- JOHANSSON C E Discusprolapsar ur et etiologiskt synpunkt Nord Med 194 36 249
- Results of myelographies with water soluble media Acta Chir Scand 1941 99 360—361
- KREGAN J J Dermalome hypalgesia associated with herniation of intervertebral disc Arch Neurol & Psychiat 1940 50 6—83
- KELLOGGREN J H Preliminary account of referred pains arising from muscle Brit M J 1938 1 39—32
- KEY C A On paraplegia depending on the ligaments of the spine Guy's Hosp Rep 1838 1 3
- KIMBERLEY A G Low back pain and sciatica, its etiology, diagnosis and treatment Sur., Gynec & Obst 193 63 193—216
- KLEINBERG S Sciatic scoliosis due to low backache Am J Surg 1923 89—94
- KNUTSSON B Electromyographic studies in the diagnosis of lumbar disc herniations Acta Orthop Scand 1949 28 290—299
- KNUTSSON B and WIERG G On surgically treated herniated intervertebral discs Acta Orthop Scand 1958 28 106—123
- KNUTSSON E Lumbar myelography with water soluble contrast in cases of disc prolapse Acta Orthop Scand 1951 20 294—302
- KOCHER T Die Verletzungen der Wirbelsäule zugleich als Beitrag zur Physiologie des menschlichen Rückenmarks Mitt Grenzgeb Med und Chir 1896 1 44—150
- KOLSTAD P og SOLFEM G H Myelografi med vannoppløselig kontrastmiddel en analyse av 497 myelografier Nord Med 1949 61 306—309
- KITTELBERG E Electromyograms in muscular disorders J Neurol Neurosurg & Psychiat 1947 10 122—128
- KITTELBERG E and PETERSEN J Muscle weakness and wasting in sciatica due to fourth lumbar or lumbosacral disc herniations J Neurosurg 1950 2 0—9
- LANBOLZY L De la sciatique et de l'atrophie musculaire qui peut la compliquer Arch gen d med 18 1 303—393 44—443 36—38
- LANGLEY J N and KATO T The physiological action of physostigmine and its action on denervated skeletal muscle J Physiol 1915 49 410—431
- LANSCH W E and FORD J T Correlation of the myelogram with clinical and operative findings in lumbar disc lesions J Bone & Joint Surg 1960 42 A 123—206
- LASELLE C Considerations sur la sciatique Arch gen d med 1864 338—350
- LASSILA Y Luftmyelographische Befunde bei Ischiassyndrom verursachen den Bandscheibenprolapsen Acta Soc Medic fenn Duodecim Ser B 194 3 3 1—9
- LEKSELL L Thorotrastmyelografi Nord Med 1943 20 243—244

- LEHARD, R. F. End result study of the intervertebral disc. *J Bone & Joint Surg* 1947 29 435—428
- LÉRY, A. et SCHAEFFER. Sciatiques et lymphocytose. La sciatique vulgaire rhumatismale est généralement d'origine radiculaire. Le rôle des canaux sacrés antérieurs. *Bull et mem Soc med d hop de Par* 1916 40 680—686
- LEWIS, T. 1ain New York: Macmillan Co 1912 122 pp
- LINDBLOM, K. Eine anatomische Studie über lumbale Zwischenwirbelscheibenprotrusionen und Zwischenwirbelscheibenbrüche in die Foramina intervertebralia hinein. *Acta Radiol* 1941 2 711—721
- Lumbar diskprolaps vid foramina intervertebralia. *Nord Med* 1942 46 357—358
- Lumbar myelography by Abrodil. *Acta Radiol* 1946 27 1—7
- LINDGREN, E. On the diagnosis of tumours of the spinal cord by the aid of gaseumyography. *Acta Chir Scand* 1933 87 303—318
- Myelography with air. *Acta psychiat et neurol* 1933 14 385—388
- LINDSEY, D. B. Electrical activity of human motor units during voluntary contraction. *Am J Physiol* 1933 114 90—99
- LINDBLAD, T. Über Ätiologie und Pathogenese der Ischias. *Acta Med Scand* 1920 33 318—350
- Über Ätiologie und Pathogenese der Ischias und Lumbago nebst einer neuen Anschauungsweise dieser Neuralgien. *Ztschr f klin Med* 1922 33 173—216
- Om ischias och myalgi problemet och därmed sammanhängande frågor om neuralgiernas natur. *Sv Läk tidn* 1937 34 457—468
- LJUNGBÄLL, M. Nerv och muskelsymptom vid deformierande arthritiser. *Sv Läk tidn* 1913 16 833—849
- LOOFBOURROW, G. N. Electromyographic evaluation of mechanical response in mammalian skeletal muscle in different conditions. *J Neurophysiol* 1948 11 153—167
- LORTAT-JACOB, L. et SABARWAL, C. Sciatique radiculaire unilatérale. *Presse med* 1901 2 633—635
- LOVE, J. G. Protruded intervertebral discs with note regarding hypertrophy of ligamentum flavum. *JAMA* 1939 113 2022—2031
- The disc factor in low back pain with or without sciatica. *J Bone & Joint Surg* 1941 23 478—481
- LOVE, J. G. and CAMI, J. D. Root pain resulting from intravertebral protrusion of intervertebral discs: diagnosis and surgical treatment. *J Bone & Joint Surg* 1953 35 786—801
- LOVE, J. G. and WALSH, M. N. Protruded intervertebral discs. *JAMA* 1938 111 336—400
- Intraspinal protrusion of intervertebral discs. *Arch Surg* 1940 40 454—484

- LOVE, J. G. and WALSH M. V. Protruded intervertebral discs. *Surg Gynec & Obst* 1943 74 40—504
- LUCHERINI T. Radiculomyelography with thorotrast. *Riv osp* 1934 24 4 3—484
- LUSCHKA H. Die Halbhgel nke des menschlichen korpers. Berlin Georg Reimer 1858 144 pp
- MACEY H. B. Clinical aspects of protruded intervertebral disk. *Arch Surg* 1940 40 433
- MACK E. W. Electromyographic observations on the postoperative disc patient. *J Neurosurg* 1951 8 463—469
- MACFENZIE J. Some points bearing on the association of sensory disorders and visceral disease. *Fram* 1893 16 321—334
- MAUMRO R. Den lumbale discsprolaps og ligamentære rodkompression. bbb Munksgaard 1942
- MARINACCI A. A. The value of electromyography in neurology. *Calif Med J* 1944 60 314—315
- Clinical electromyography. *Calif San Lucas Press* 1955
- The use of electromyography in the differential diagnosis of lumbar herniated disks. *Bull Los Angeles Neurol Soc* 1958 23 60—1
- MAYERDING H. W. Spondylolisthesis as an etiologic factor in backache. *JAMA* 1938 111 1971—196
- Low backache and sciatic pain associated with spondylolisthesis and protruded intervertebral disc: incidence, significance and treatment. *J Bone & Joint Surg* 1941 23 461—466
- MENDELSON R. A. and SOLA A. Electromyography in herniated lumbar disks. *A M A Arch Neur Psychiat* 1958 9 142—145
- MIDDLETON G. S. and TEACHER J. H. Injury of the spinal cord due to ruptures of an intervertebral disc. *Glasgow Med J* 1911 6 1—6
- MIXTER W. J. and BARR J. S. Rupture of intervertebral disc with involvement of spinal canal. *New England J Med* 1931 211 210—215
- NAFFZIGER H. C. INMAN V. and SALADERS I. B. Lesions of the intervertebral disc and lig flava. *Surg Gynec & Obst* 1938 68 288—299
- NICHOLS B. H. and NOSIC W. A. Myelography with the use of thorium dioxide solution (thorotrast) as a contrast medium. *Radiol* 1940 34 2—466
- NORDEHOFT J. M. Om myelografi med abrodil. *Nord Med* 1944 21 2 2—95
- NORLÉN G. On the value of the neurological symptoms in sciatica for the localization of a lumbar disc herniation. *Acta Chir Scand* 1944 91 suppl 95
- Om behandling av artrosis deformans coxae med rotarskarning. *Sv Lak tidn* 1944 41 661—665
- NOTTA A. Memoire sur les lesions fonctionelles qui sont sous la dependance des névralgies. *Arch gen d med* 1854 9 552—555

- LINARD R E End result study of the intervertebral disc *J Bone & Joint Surg* 1947 29 425—428
- LEPÉ A et SCHAEFFER Sciatiques et lymphocytose La sciatique vulgaire rhumatismale est généralement d'origine radiculaire Le rôle des canaux sacrés inférieurs *Bull et mem Soc med d hop de Lar* 1916 40 680—686
- LEWIS T Pain New York Macmillan Co 1942 192 pp
- LINDBLÖM K Eine anatomische Studie über lumbale Zwischenwirbelscheibenprotrusionen und Zwischenwirbelscheibenbrüche in die Foramina intervertebralia hinein *Acta Radiol* 1941 22 711—721
- Lumbala diskprolaps vid foramina intervertebralia *Nord Med* 1949 16 3557—3598
- Lumbar myelography by Arödi *Acta Radiol* 1946 27 1—7
- LINDEREN L On the diagnosis of tumours of the spinal cord by the aid of myelography *Acta Chir Scand* 1939 82 303—318
- Myelography with air *Acta psychiat et neurol* 1939 11 385—388
- LINDSLEY D B Electrical activity of human motor units during voluntary contraction *Am J Physiol* 1935 114 90—99
- LINDBLÖM F Über Ätiologie und Pathogenese der Ischias *Acta Med Scand* 1940 83 318—380
- Über Ätiologie und Pathogenese der Ischias und Lumbago nebst einer neuen Anschauungsweise dieser Neuralgien *Ztschr f klin Med* 1942 93 173—216
- Om ischias och myalgiproblemet och därmed sammanhängande frågor om neuralgiernas natur *Sv Läk tidn* 1937 31 457—468
- LJUNGBÄHL M Nerv och muskelsymptom vid deformerande arthrit *Sv Läk tidn* 1919 16 833—843
- LOOFBOURNOW G N Electromyographic evaluation of mechanical response in mammalian skeletal muscle in different conditions *J Neurophysiol* 1948 11 153—167
- LORTAT JACOB I et SABARÉAU G Sciatique radiculaire unilatérale *Presse med* 1904 2 633—635
- LOVE J G Protruded intervertebral discs with note regarding hypertrophy of ligamentum flavum *JAMA* 1939 113 2023—2034
- The disc factor in low back pain with or without sciatica *J Bone & Joint Surg* 1947 29 438—447
- LOVE J G and CAMP J D Root pain resulting from intraspinal protrusion of intervertebral discs diagnosis and surgical treatment *J Bone & Joint Surg* 1934 16 6—804
- LOVE J G and WALSH M N Protruded intervertebral discs *JAMA* 1938 111 396—400
- Intraspinal protrusion of intervertebral discs *Arch Surg* 1940 40 424—481

- VON REIS G SAHLGREN E and SJÖQVIST O En operationsmetod vid arthrosis deformans coxae Nord Med 1944 21 557—559
- ROGER H Les sciatiques formes cliniques et traitement des sciatiques rhumatismales Rev neurol 1930 1 1037—1096
- ROGERS M H Sciatica etiology and treatment Boston M & S J 1911 165 60—69
- ROMBERG M H Lehrbuch der Nervenkrankheiten Berlin Alexander Duncker 1853 I I 327 pp
- ROSS P and JELMAN F Postoperative analysis of 366 consecutive cases of herniated lumbar discs Am J Surg 1957 84 657—662
- ROUSSET 1804 quoted by Dubarry
- RÖVIG G Rupture of lumbar discs with intraspinal protrusion of the nucleus pulposus Acta Chir Scand 1949 suppl 144
- SAHLGREN E Beror ischias alltid på lokal affektion av rot eller nerv? Sv Lak tidn 1947 39 1137—1146
- Nye synpunkter på lumbago ischiasproblemet Nord Med 1946 29 0,
- SASHIN D Critical analysis of anatomy and pathologic changes in sacro iliac joints J Bone & Joint Surg 1930 12 891—910
- SCHIFF M Über motorische Lähmung der Zunge Arch f Physiol Heilkunde 1851 10 79—113
- SCHMORL G Über die an den Wirbelbandscheiben vorkommenden Ausdehnungs- und Zerreissungsvorgänge und die dadurch an ihnen und der Wirbelspongiosa hervorgerufenen Veränderungen Verhandl d deutsch path Gesellsch 1927 22 250—262
- Über Knorpelknötchen an der Hinterfläche der Wirbelbandscheiben Fortschr Röntgenstr 1933 40 629—634
- SCHMORL G und JUNGHAUS H Die gesunde und kranke Wirbelsäule im Röntgenbilde Leipzig Georg Thieme 1932
- SCHOBER P Kritische Betrachtungen über das Laseguesche Zeichen und die sogenannte Ischias Deutsche med Wchnschr 1910 66 1269—1271
- SCOTT W G and FLEWELL T Myelography with pantopaque and a new technique for its removal Radiol 1914 43 241—243
- SINNING Å och SJÖQVIST O Senresultaten vid diskbräck En efterundersökning av 400 opererade fall Nord Med 1947 34 1178—1179
- DE SOLLE S Sciaticque basale et disques lombo sacres Presse méd 1940 48 5 0—5 1
- SHEA P A and WOODS W W Electromyography as an aid in clinical diagnosis A M A Arch Int Med 1952 96 187—193
- The diagnostic value of the electromyograph Brit J Phys Med 1956 1 febr 1—8
- SHEA P A WOODS W W and WERDEN D H Electromyography in

- diagnosis of nerve root compression syndrome Arch Neurol & Psychiat 1950 64 93—104
- SHERINGTON C S Note on the knee jerk and the correlation of action of antagonistic muscles Proc Roy Soc 1893 52 56
- SHINNERS B M and HAMBY W B The results of surgical removal of protruded lumbar intervertebral discs J Neurosurg 1944 1 117—122
- SICARD J A Névroses et funiculites vertebrales Presse med 1918 26 9—11
- SICARD J A et FORSTIER J L Methode radiographique d'exploration de la cavité epidurale par le lipiodol Rev neurol Par 1921 28 1264—1266
- SJÖQVIST O De lumbala diskbräckens klinisk diagnos samt exstirpation utan laminectomi Nord Med 1913 13 687—693
- SMITH A DE F Posterior displacement of fifth lumbar vertebra J Bone & Joint Surg 1931 16 847—858
- SMITH PETERSEN M N and ROGER W A End result study of arthrodesis of the sacro iliac joint for arthritis traumatic and nontraumatic J Bone & Joint Surg 1956 8 118—131
- SPLRLING R G and BRADFORD F K Neurologic aspects of herniated nucleus pulposus at the fourth and fifth lumbar interspace JAMA 1939 113 2013—2022
- SPLRLING R G and GRANTHAM L G Neurologic picture of herniations of nucleus pulposus in lower part of lumbar region Arch Surg 1940 40 345—358
- SPLRLING R G MAYFIELD I H and ROGERS J B Hypertrophy of ligamenta flava as cause of low back pain JAMA 1937 109 28—33
- STAINDLER A Interpretation of sciatic radiation and syndrome of low back pain J Bone & Joint Surg 1940 22 28—34
- STENSTRÖM R Widening of the root defect in lumbar myelogram by abrodil Acta Radiol 1948 29 303
- STENSTRÖM T Eine klinische Studie über Neuritis Symptome bei Ischias Acta psychiat et neurol 1931 6 593—644
- STERNBERG 1893 quoted by Norlen
- STERNBERG H Über Wurzelschias Munchen med Wchnschr 1910 27 17 6—1,80
- STÄHL F Clinical diagnosis of lumbar disc herniations Acta Orthop Scand 1949 18 141—153
- TOWNE E B and REICHERT F I Compression of lumbosacral roots of spinal cord by thickened ligamenta flava Ann Surg 1931 91 327—336
- WAGNER L C Congenital defects of lumbosacral joints with associated nerve symptoms Am J Surg 1935 27 311—327
- WALK L Slight myelographic deformities in sciatica Acta Radiol 1958 20 226—229

- VALLEIN F L I *Traité des neuralgies* Paris J B Bailliere 1841 480—647
- WARIS W Lumbar disc herniation *Acta Chir Scand* 1949 suppl 140
- WEDDELL G FEINSTEIN B and PATTLE R L *The electrical activity of voluntary muscle in man under normal and pathological conditions* Brain 1944 67 178—257
- WERTHEIM SALOMONSON J h A Neuralgie und Myalgie In Lewandowsky *Handbuch der Neurologie* 1911 2 1—50
- WIBERG G Prolaps av intervertebraldiskerna som orsak till ischias Uppsala läkaref förh 1943 48 415—431
- WILLIAMS I C Reduced lumbosacral joint space its relation to sciatic irritation JAMA 1932 99 1667—1689
- WÖRINGER E THOMASKE G und BALMGARTNER J Die Myelographie mit wasserlöslichem Kontrastmittel in der Diagnose des lumbosacralen Bandscheibenprolapses Bericht über 600 Fälle Nervenarzt 1956 27 54—59
- YASKIN C and FINKELSTEIN A Low back and leg pains clinical considerations Clinics 1944 3 261—308
- YEOHAN W Relation of arthritis of sacro iliac joint to sciatica Lancet 1928 1119—1129
- YOUNG B B and SCOTT M Air myelography substitution of air for lipiodol in roentgen visualization of tumours and other structures in spinal canal Am J Roentg 1938 39 18—192
- ÅBERBLOM N A Bidrag till diskussion om behandling av ischias Nord tidsskr f terap 1908 7 148—151

U S MEDICAL COL  
LIBRARY, J6

S M S MEDICAL COLLEGE LIBRARY  
DUE DATE SLIP

*This book is to be returned on or before the date  
marked below —*

A fine of 25 nP will be charged for each day  
the book is kept overtime

|  |  |  |
|--|--|--|
|  |  |  |
|--|--|--|



FROM THE ORTHOP CLINIC, UNIVERSITY OF LUND  
(CHIEF PROF G WIBERG) AND THE DEPARTMENT OF MEDICAL  
PHYSICS (HEAD ASS PROF P PETERSEN) OF THE INSTITUTE OF  
PHYSIOLOGY LUND (CHIEF PROF G KAHLSON)

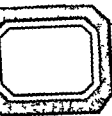
# EXPERIMENTAL INVESTIGATION OF CORROSION OF STAINLESS STEELS USED IN BONE SURGERY

BY

HANS EMNÉUS



EJNAR MUNKSGAARD  
COPENHAGEN 1961





FROM THE ORTHOP CLINIC, UNIVERSITY OF LUND  
(CHIEF PROF G WIBERG) AND THE DEPARTMENT OF MEDICAL  
PHYSICS (HEAD ASS. PROF P PETERSEN) OF THE INSTITUTE OF  
PHYSIOLOGY LUND (CHIEF PROF G KAHLSON)

EXPERIMENTAL INVESTIGATION OF CORROSION  
OF STAINLESS STEELS USED  
IN BONE SURGERY

BY

HANS EMNÉUS



EJNAR MUNKSGAARD  
COPENHAGEN 1961



ACTA ORTHOPAEDICA SCANDINAVICA

SUPPLEMENTUM no XLIVb

---

FROM THE ORTHOP CLINIC UNIVERSITY OF LUND  
(CHIEF PROF G WIERC) AND THE DEPARTMENT OF MEDICAL  
PHYSICS (HEAD ASS PROF P FLTERSEN) OF THE INSTITUTE OF  
PHYSIOLOGY LUND (CHIEF PROF G KARLSON)

EXPRIMENTAL INVESTIGATION OF CORROSION  
OF STAINLESS STEELS USED  
IN BONE SURGERY

BY

HANS EMNEUS



EJNAR MUNKSGAARD  
COPENHAGEN 1961

*Printed in Sweden*

HÅKAN GILLSSONS BOKTRYCKERI

## INTRODUCTION

A stainless steel may be noble enough for some purposes in certain chemical environments. On introduction of a new component to the material or the environment however the relative nobility of the metal may prove insufficient and the steel can then no longer be regarded as stainless for its purpose.

An attack of various corroding agents on stainless steel may result in

- 1 General surface corrosion
- 2 Pitting
- 3 Intergranular corrosion
- 4 Crevice corrosion
- 5 Contact or galvanic corrosion
- 6 Stress corrosion

As a rule the chromium nickel steels of 18/8 type do not undergo general corrosion in chloride solution at pH 5—8. Intergranular corrosion can only occur on material which has been sensitized at 500—800° C. There is always a risk of stress corrosion in the presence of both chlorides and oxygen provided that the stainless steel is in a state of tension. At such a low temperature as 40° C and at such a concentration of NaCl as in body fluids the risk is probably negligible. The other condition tension is present for example in cerclage (residual tension) where the tension is of sufficient order. Since the first condition for stress corrosion — high chloride concentration and high temperature — is not present this type of corrosion can be excluded in surgical practice (BERG — personal comm.)

As far as *surgical appliances* are concerned three types of

corrosion must be considered pitting, crevice corrosion and galvanic corrosion. It should be emphasized once more that surgical cold working, e.g. twisting and cutting of wires (e.g. etc.) of chromium nickel steel and chromium nickel molybdenum steel is a causal factor of pitting and crevice corrosion (See LAMNUS 1960)

## ENVIRONMENTS

The environments of a stainless steel appliance are important. Highly concentrated solution of electrolytes favour corrosion. Chlorides are broadly speaking the strongest corrosives of these. Other anions (e.g. ureates, lactates, sulphates, phosphates, oxalates, gluconates and fatty acid salts) occur in such low concentrations in living tissue and have such a relatively slight accelerating effect on corrosion that their effect is negligible compared with that of the aggressive chlorides (FINK & SMATKO 1948). In damaged biological tissue the pH probably varies relatively much. The pH of recently damaged tissue may be 5.3—5.6 after which it rises to 7.3 during healing (LIRUSON 1959).

UHLIG (1948) has shown pitting to be most pronounced between pH 5 and pH 9 in chloride solutions. There is little difference in the corrosive effect of a chloride solution at pH 5 and pH 7 (see diagram 1). This is in line with the investigations of FINK & SMATKO (1948). They found no difference between the destructive effects of physiological saline, ordinary serum, serum acidified to pH 5—pH 5.5 with the  $\text{KH}_2\text{PO}_4$  serum containing sulphonamide and serum inoculated with *Staphylococcus aureus*.\*

Stainless steels tend to become passive i.e. to be covered with a film of chromium oxides and entrapped oxygen in the presence of oxygen e.g. in oxygen containing solutions. Such a film offers considerable protection against most corrosive

---

\* Observe that phosphate and amines are inhibitors of pitting under certain conditions (remark of the author)



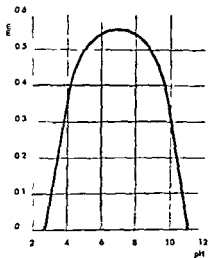


Diagram 1

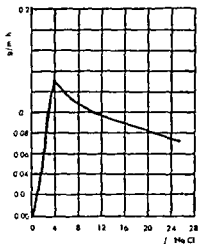


Diagram 2

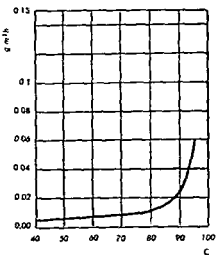


Diagram 3

Diagram 1 Influence of pH on the depth of pitting in 18-8-steel in NaCl

Uhlir, 1948

Diagram 2 Influence of concentration of NaCl on pitting in 18-8 steel

Uhlir, 1948

Diagram 3 Influence of temperature on pitting in 18-8 steel in 1% NaCl

Uhlir, 1948

agents with the exception however of halides particularly chlorides. If a passive film is attacked by chlorides for example the attack will be pitting; the individual points will be active or anodic and be extremely small in relation to the passive surface. The current density will therefore be high and corrosion will progress rapidly at such points. The result will be pitting corrosion. The presence of oxidising agents e.g. oxygen in the solution will accelerate this attack still more by depolarization of the cathode. Therefore living tissue with its relatively high chloride content and oxygen content constitutes a particularly stimulating medium for pitting. Sea water with NaCl in a concentration of 3 per cent is certainly more aggressive than living tissue and the aggressiveness is proportional to the concentration of the chloride (LILIE 1918). See diagram 2.

## PREVIOUS INVESTIGATIONS

Studies of previous surgical alloys and elements used during the 1920ies and 1930ies in the manufacture of surgical appliances have been surveyed by HENSCHE & GERLACH (1941) HÖRDIS JÖRGENSEN (1941) and VENABLE & STUCK (1947). These surveys are however of less relevance to the present investigation. JONES & FRIEDMAN (1936) compared the behaviour of chromium-vanadium steel and chromium-nickel steel in physiological saline. They found loss of weight of chromium-vanadium steel but not of chromium-nickel steel. VENABLE & STUCK (1947) compared chromium steel, chromium-nickel steel (18-8) and vitallium. The samples were mounted in the form of plates with screws in screw holes and the corrosive medium was physiological saline. They analyzed the electrolytes after 3 weeks and found them to contain corrosion products of chromium steel and of chromium-nickel steel respectively. No corrosion products were found in the corrosive medium around vitallium.

LINK & SMATKO (1918) compared the losses in weight in physiological saline and serum of chromium-nickel steel

chromium nickel molybdenum steel and vitallium and found the loss of weight to be the smallest for chromium nickel steel (AISI 302) <sup>1</sup>

CLARK & HICKMAN (1933) studied the corrosion tendency of conventional surgical alloys by measuring the so-called back EMF. From the back EMF it is however difficult to obtain detailed information about the behaviour of the alloys after they have been cold worked or about the weaknesses they may have when used for special purposes. BOWDEN, WILLIAMSSON & LAING (1933) studied changes occurring during application of an appliance and found a transfer of substance from the tool (screw driver) to the screw head. They also mentioned the deformation of the screw head. But they did not investigate these two effects of cold working separately. EMNELS & PETERSEN (1938) studied the effect of cold working of annealed 18/8 wire as judged by measurements of the potentials.

A fairly thorough search of the literature failed to reveal any investigation in which the corrosive conditions were studied experimentally *in vitro* with respect to the entire situation prevailing on insertion of different types of appliances (screw plates and wires).

VENABLE & STUCK (1947) as well as FINE & SMATKO (1948) were unable to ascertain under what conditions and where corrosion occurred. In addition it is difficult to accept the results of FINE & SMATKO who found corrosion of vitallium to be more common than that of 18/8 steels.

Experimental investigations have been made of the reaction of the tissue around various metal implants, generally around single implants (VENABLE & STUCK 1937; HJ. JØRGENSEN

---

<sup>1</sup> Sometimes the passive surface is intact despite strongly corrosive environments. This may be due to the surface treatment of the material, to chance, to variation in the composition of the material and to the mechanical stress being only relatively slight. Under such favourable circumstances AISI 302 may be resistant even under conditions in which vitallium for example is attacked. But it should be emphasized that this is very rare.

1941 BOTTIE & DAVENPORT 1940 LEVINTHAL 1951 VOIGT & SEYFARTH 1955 and (LARK & HICKMAN 1958) and around appliances such as plate and screws used in osteosynthesis (HEDGACK 1940 KEY 1941 and 1946 BLUNT HEDGACK & MURRAY 1952 and JERGENSEN 1955) or around metals worked in some other way (FAIRC *et al* 1955 and 1958 and LAMMUS & STENRAM 1960). The investigations suggest that corrosion is liable to occur at the points of contact between the same or different kinds of metals (possibly crevice or galvanic corrosion)<sup>1</sup> (BLUNT HEDGACK MURRAY 1952 JERGENSEN 1955 and LAMMUS & STENRAM 1960). In addition the site at which the metal has been surgically cold worked also favours corrosion (FAIRC *et al* 1958 and LAMMUS & PITKINEN 1958).

There is every reason to consider the corrosion problem mainly from a biological standpoint. In the choice of material for technical constructions to be used in salt water the manufacturer can often select a material which is not fully resistant and which can be protected by means of cathodic protection i.e. making the metal the anode in a D.C. circuit. The cathodic material is then not attacked. This is in principle what occurs when iron is covered with or connected to zinc. Zinc is less noble than iron and therefore it becomes the anode and the iron the cathode. Zinc plated surfaces are therefore not attacked until the zinc has been consumed. In technical constructions a continuous supply of metal ions from the construction to the environment is as a rule less important as long as the attack is not punctate or sufficient to jeopardize the strength of the construction.

Technical problems arising from exposure of a construction to salt water can be solved by using highly alloyed Cr-Ni-Mo steel and by giving the construction a shape and surface tending to minimize the effect of corrosion. This is however only sometimes possible in the design of surgical appliances.

When the surgeon has inserted the nails and/or applied the cerclage wires or plates with screws it is not possible to treat

the surface of the inserted construction which must therefore remain unchanged in the corrosive environments. Neither can he arrange any cathodic protection. Nor can he tolerate resolution of more than minute amounts of metal ions because of their irritating action.

## PERSONAL INVESTIGATION

### Methods and Material

PETERSEN & EMNEUS (1960) showed that their modification of the ferroxy test is particularly suitable for qualitative studies of corrosion of surgical appliances.<sup>1</sup> PIER & SCHWENK (1960) studied pitting on 18/8 steel with the ferroxy test. EMNEUS (1960) inserted metal implants into chickens. Three months later the implants were removed and studied for corrosion by the ferroxy test. He found fair agreement between the amount of iron pigment in the tissue and the results of subsequent ferroxy test of the steel implants. He also found that the ferroxy test placed higher demands on the metal than biological tests. In addition EMNEUS & PETERSEN showed that the ferroxy test can demonstrate corrosion even of cobalt and nickel alloys. EMNEUS (1960) compared the reaction of the tissue as demonstrated by an accumulation of metal pigments with the ferroxy test of cobalt alloys and found some agreement though the cobalt alloys appeared to be resistant to the otherwise provocative effect of ferroxy test.

In order to check to what extent the result of the ferroxy test varies with the salt solution (concentration of NaCl) to which the test objects were exposed the following experiments were performed.

0.9 mm annealed 18/8 wire (832 M) of the type used for cerclage was cut into 600 pieces about 1 cm long. The cutting process which is a type of cold working deforms the ends of these 1 cm long pieces. The ends are then liable to undergo

---

All experiments were begun at 40° C and proceeded at room temperature (note diagram 3).

1911 BOTHL & DAVERNORT 1910 LIVENHAL 1951 VOIGT & SEYFART 1955 and (LARK & HICKMAN 1958) and around appliances such as plate and screws used in osteosynthesis (HUDACK 1910 KEY 1911 and 1916 BLUNT HUDACK & MURRAY 1952 and JERGLSLN 1955) or around metals worked in some other way (LAINE *et al* 1955 and 1958 and I MINUS & STENRAM 1960). The investigations suggest that corrosion is liable to occur at the points of contact between the same or different kinds of metals (possibly crevice or galvanic corrosion)<sup>1</sup> (BLUNT HARDOCK MURRAY 1952 JERGLSLN 1955 and I MINUS & STENRAM 1960). In addition the site at which the metal has been surgically cold worked also favours corrosion (LAINE *et al* 1958 and I MINUS & PITIRSI 1958).

There is every reason to consider the corrosion problem mainly from a biological standpoint. In the choice of material for technical constructions to be used in salt water the manufacturer can often select a material which is not fully resistant and which can be protected by means of cathodic protection, i.e. making the metal a cathode in a D.C. circuit. The cathodic material is then not attacked. This is in principle what occurs when iron is covered with or connected to zinc. Zinc is less noble than iron and therefore it becomes the anode and the iron the cathode. Zinc plated surfaces are therefore not attacked until the zinc has been consumed. In technical constructions a continuous supply of metal ions from the construction to the environment is as a rule less important, is longer, as the attack is not punctate or sufficient to jeopardize the strength of the construction.

Technical problems arising from exposure of a construction to salt water can be solved by using high alloyed Cr-Ni-Mo steel and by giving the construction a shape and surface tending to minimize the effect of corrosion. This is however only sometimes possible in the design of surgical appliances.

When the surgeon has inserted the nails and/or applied the cretalic wires or plates with screws it is not possible to treat

---

<sup>1</sup>Remark of the author

the surface of the inserted construction which must therefore remain unchanged in the corrosive environments. Neither can he arrange any cathodic protection. Nor can he tolerate resolution of more than minute amounts of metal ions because of their irritating action.

## PERSONAL INVESTIGATION

### Methods and Material

PETERSEN & EMNELS (1960) showed that their modification of the ferroxyl test is particularly suitable for qualitative studies of corrosion of surgical appliances.<sup>1</sup> PIER & SCHWENK (1960) studied pitting on 18/8 steel with the ferroxyl test. EMNELS (1960) inserted metal implants into chickens. Three months later the implants were removed and studied for corrosion by the ferroxyl test. He found fair agreement between the amount of iron pigment in the tissue and the results of subsequent ferroxyl test of the steel implants. He also found that the ferroxyl test placed higher demands on the metal than biological tests. In addition EMNELS & PETERSEN showed that the ferroxyl test can demonstrate corrosion even of cobalt and nickel alloys. EMNELS (1960) compared the reaction of the tissue as demonstrated by an accumulation of metal pigments with the ferroxyl test of cobalt alloys and found some agreement though the cobalt alloys appeared to be resistant to the otherwise provocative effect of ferroxyl test.

In order to check to what extent the result of the ferroxyl test varies with the salt solution (concentration of NaCl) to which the test objects were exposed the following experiments were performed.

0.9 mm annealed 18/8 wire (832 M) of the type used for cerclage was cut into 600 pieces about 1 cm long. The cutting process which is a type of cold working deforms the ends of these 1 cm long pieces. The ends are then liable to undergo

---

All experiments were begun at 40°C and proceeded at room temperature (note diagram 3).

corrosion because the passive surface layer is destroyed or damaged there. There is hardly any transfer of the metal from the piers to the wire though metal from the wire may be transferred to the piers. The 600 pieces of wire were divided into 6 groups of 100 each and deposited in three concentrations of NaCl and in 2 concentrations of indicators ( $\text{K}_2\text{Fe}(\text{N})_6$ ) 2 per cent and 0.2 per cent respectively — Table 1.

TABLE 1

| Concentration of NaCl | Concentration of $\text{K}_2\text{Fe}(\text{N})_6$ | Cast on is with indication of corrosion |
|-----------------------|--|---|
| 0.1 %                 | 0.2 %  | 12/200                                  |
| 0.30 %                | 0.2 %  | 33/200                                  |
| 3.0 %                 | 0.2 %  | 100/200                                 |
| 0.1 %                 | 2.0 %  | 120/200                                 |
| 0.30 %                | 2.0 %  | 127/200                                 |
| 3.0 %                 | 2.0 %  | 150/200                                 |

It is clear from the table that the tendency to corrosion increased with increasing concentration of the NaCl from 0.1 % up to 3 per cent. It was decided to use 0.9 per cent NaCl.

Comparative tests were made with 0.9 % NaCl solution and human plasma and dog plasma. The effect of plasma is more difficult to judge owing to deposition of protein and fat on the wire specimens but despite this it was obvious that the tendency to corrosion was greater in 0.9 % NaCl solution than in plasma.

2 mm 18-8 wire used for the test was almost always severely damaged or deformed when cut into pieces and practically all the pieces showed corrosion when deposited in 0.9 % NaCl solution but only 70 per cent of those placed in plasma.

Since the values obtained in most of the experiments described below were relative it was decided to limit the experiment to 0.9 % NaCl solution.

To summarize the ferroxyl test is provocative as pointed out previously by PETERSEN & JENSEN (1960) and KETZELNIC



(1960) This provocative effect increases with the concentration of the indicator. A chloride ion concentration corresponding to 0.9 per cent NaCl is slightly higher than that in the body but was used for all experiments. ENVELUS (1960) showed that the ferroxyl test in 0.9 per cent NaCl gives results agreeing well with biological tests and that NaCl could at any rate be used for the purpose under consideration. Plasma is perhaps less aggressive than 0.9 per cent NaCl but it has several disadvantages if it is to be used in a qualitative corrosion test such as the ferroxyl test. When comparing the corrosion tendency of stainless steels for surgical appliances it is quite justified to place higher demands on such metals than might be required by living tissue.

### EXPERIMENTS WITH COLD WORKING OF ANNEALED STAINLESS STEEL WIRE OF 3 QUALITIES

Cerclage wire used in Sweden usually consists of stainless steel wire of 18.8 type. This quality is called 2R2 (Särviken) and 832 M (Avesta). When cold worked as when used for cerclage the twisted segment becomes harder than the eye (ENVELUS & PETERSEN 1958). In addition the passive layer will be damaged at the free cut ends.

Modern appliances for osteosynthesis material of steel is usually made of 1.4301 MB or AISI 316 (Table 2) if manufactured in England or America. If manufactured in Sweden they will be made of 453 S and 832 SV or even sometimes of chromium steel 1 MB and AISI 316 correspond approximately to Avesta 832 Sk. FST of Särviken 2R2 or Avesta 832 M.

AISI 317 and Avesta 832 SL were not placed on the market until some years ago.

In the experiments 12.5 cm long electropolished wires 1 mm in diameter and of the three qualities 2R2, 832 Sk and 832 SL were twisted a definite number of turns in a special apparatus the stress in the wire increasing per number of turns (see Fig. 1).

TABLE 2

|  | C         | Cr      | Ni    | Mo  | Ce  |
|--|-----------|---------|-------|-----|-----|
| 1ST <sup>1</sup> AISI 303 <sup>1</sup> R <sup>2</sup><br>and 83 <sup>3</sup> MP <sup>4</sup> | 0.05-0.10 | 17.5-18 | 8-9.5 | —   | Bal |
| 832 SV <sup>3</sup>  | 0.05      | 17.5    | 10.0  | 1.5 |     |
| 1MB <sup>1</sup> AISI 316 <sup>1</sup> 83 <sup>3</sup><br>SK <sup>4</sup>                    | 0.05-0.08 | 17.5-18 | 8-11  | 2.7 |     |
| 153 S <sup>3</sup>   | 0.08      | 16      | 5     | 1.5 |     |
| AISI 317 <sup>1</sup>  | 0.05-0.08 | 17.5    | 11    | 3.7 |     |
| 83 SL <sup>3</sup>   | 0.05      | 17.5    | 11-1  | 4.5 |     |

<sup>1</sup> Eirth Vickers Great Britain

<sup>2</sup> Sandvikens Jernverks AB Sweden

<sup>3</sup> Avesta Jernverks AB Sweden

<sup>4</sup> American Iron and Steel Institute specifications

Table 2 gives the approximate compositions of some representative chromium nickel and chromium nickel molybdenum steels. Steels of the types may be represented in surgical appliances used in Sweden today.

The steel qualities used in the present investigation are italicized.

For this experiment 50 pieces of cerclage wire of the three different qualities each were turned 4 times and 100 pieces of wire of each quality were cut off with pliers. The twisted pieces of cerclage wire were removed from the apparatus before they were cut off. In order to prevent corrosion of the cut ends the latter were painted with cellulose lacquer. The hundred 1 cm long pieces referred to above were studied for corrosion at the cut ends.

Twenty five pieces of twisted wire and 50 pieces of wire cut from every quality were deposited in ferroxyl<sup>®</sup> with an indicator concentration of 0.2 per cent. The other series of 25 and 50 pieces respectively were deposited in ferroxyl<sup>®</sup> with an indicator concentration of 2 per cent. They were studied for corrosion after 24 hours. See Table 3 and Figs. 2a, 2b, 2c and 2d.

In clinical work the cerclage wire must be twisted at least 4 times and if it should be twisted as lightly as possible which

TABLE 3

| Quality | Concentration of electrolyte | Concentration of indicator | Twisted parts with indication of corrosion | Cut ends with indication of corrosion |
|---------|------------------------------|----------------------------|--|---------------------------------------|
| 2R2     | 0.90% NaCl                   | 0.2%                       | 24.2%                                      | 34.100                                |
| 2R2     |                              | 2.0                        | 24.2%                                      | 26.100                                |
| 832 Sh  |                              | 0.2                        | 24.2%                                      | 53.100                                |
| 832 Sh  |                              | 2.0%                       | 25.2%                                      | 62.100                                |
| 832 SL  |                              | 0.2%                       | 0.2%                                       | 14.100                                |
| 832 SL  |                              | 2.0                        | 0.2%                                       | 11.100                                |

TABLE 4

| Quality | Concentration of electrolyte | Concentration of indicator | Number of twists | Twisted parts with indication of corrosion |
|---------|------------------------------|----------------------------|------------------|--|
| 2R2     | 0.90% NaCl                   | 2.0%                       | 3                | 25.2%                                      |
| 832 Sh  |                              |                            | 3                | 24.2%                                      |
| 832 SL  |                              |                            | 6                | 0.2%                                       |

in the specially devised apparatus used in the present investigation was 6 times for a length of 12.5 cm. If twisted 7—8 times the wire telescopes or breaks.

The experiment was extended to include tests with a reduction of the requirements placed on 2R2 and 832 Sh in that the (25 pieces) wire was twisted only three times (Fig. 2b). 25 pieces of 832 SL were twisted 6 times (Fig. 2d). As before the free ends were painted with lacquer. The experiment was otherwise the same as before (Table 4).

### Results

Judging from the ferroxy test 1 mm wire of 2R2 and 832 Sh does not tolerate the stress involved by cerclage. 832 SL on the other hand tolerates almost maximal working of this type. Cutting with the pliers however caused corrosion in the free ends of 832 SL though only occasionally.

TABLE 2

|  | C         | Cr    | Ni    | Mo  | Ce  |
|--|-----------|-------|-------|-----|-----|
| 1ST <sup>1</sup> AISI 303 <sup>2</sup> R <sup>3</sup><br>an 132 M <sup>4</sup> | 0.05-0.10 | 17-18 | 8-11  | —   | Hal |
| 8J2 S <sup>2</sup>   | 0.05      | 17    | 10.0  | 1.5 |     |
| 1MB <sup>1</sup> AISI 316 <sup>2</sup> 8J<br>SK <sup>2</sup>                   | 0.0-0.05  | 17-18 | 8-11  | 2.7 |     |
| 4J2 S <sup>2</sup>   | 0.05      | 21    | 5     | 1.5 |     |
| AISI 317 <sup>4</sup>  | 0.05-0.08 | 17-5  | 14    | 3.7 |     |
| 8J SK <sup>2</sup>   | 0.05      | 17-5  | 14-15 | 4   |     |

<sup>1</sup> Lärth Vickers Great Britain

<sup>2</sup> Sandvikens Jernverks AB Sweden

<sup>3</sup> Avesta Jernverks AB Sweden

<sup>4</sup> American Iron and Steel Institute specifications

Table 2 gives the approximate compositions of some representative chromium nickel and chromium nickel molybdenum steels. Steels of these types may be represented in surgical appliances used in Sweden today.

The steel qualities used in the present investigation are indicated

For this experiment 50 pieces of cerclage wire of the three different qualities each were turned 3 times and 100 pieces of wire of each quality were cut off with pliers. The twisted pieces of cerclage wire were removed from the apparatus before they were cut off. In order to prevent corrosion of the cut ends the latter were painted with cellulose lacquer. The hundred 1 cm long pieces referred to above were studied for corrosion at the cut ends.

Twenty five pieces of twisted wire and 50 pieces of wire cut from every quality were deposited in ferroxyl gel with an indicator concentration of 0.2 per cent. The other series of 25 and 50 pieces respectively were deposited in ferroxyl gel with an indicator concentration of 2 per cent. They were studied for corrosion after 24 hours. See Table 3 and Figs 2a, 2b, 2c and 2d.

In clinical work the cerclage wire must be twisted at least 4 times and if it should be twisted as tightly as possible which

TABLE 3

| Quality | Concentration of electrolyte | Concentration of indicator | Twisted parts with indication of corrosion | Cut ends with indication of corrosion |
|---------|------------------------------|----------------------------|--|---------------------------------------|
| 2R2     | 0.90% NaCl                   | 0.2%                       | 24/25                                      | 34/100                                |
| 2R2     |                              | 2.0%                       | 24/25                                      | 76/100                                |
| 832 Sh  |                              | 0.2%                       | 24/25                                      | 53/100                                |
| 832 Sh  |                              | 2.0                        | 25/25                                      | 67/100                                |
| 832 SL  |                              | 0.2%                       | 0/25                                       | 14/100                                |
| 832 SL  |                              | 2.0                        | 0/25                                       | 11/100                                |

TABLE 4

| Quality | Concentration of electrolyte | Concentration of indicator | Number of twists | Twisted parts with indication of corrosion |
|---------|------------------------------|----------------------------|------------------|--|
| 2R2     | 0.90% NaCl                   | 2.0%                       | 3                | 25/25                                      |
| 832 Sh  |                              |                            | 3                | 24/25                                      |
| 832 SL  |                              |                            | 6                | 0/25                                       |

in the specially devised apparatus used in the present investigation was 6 times for a length of 12.5 cm. If twisted 7—8 times the wire telescopes or breaks.

The experiment was extended to include tests with a reduction of the requirements placed on 2R2 and 832 Sh in that the (25 pieces) wire was twisted only three times (Fig. 2b). 25 pieces of 832 SL were twisted 6 times (Fig. 2d). As before the free ends were painted with lacquer. The experiment was otherwise the same as before (Table 4).

### Results

Judging from the ferroxy test 1 mm wire of 2R2 and 832 Sh does not tolerate the stress involved by cerclage. 832 SL on the other hand tolerates almost maximal working of this type. Cutting with the pliers however caused corrosion in the free ends of 832 SL though only occasionally.

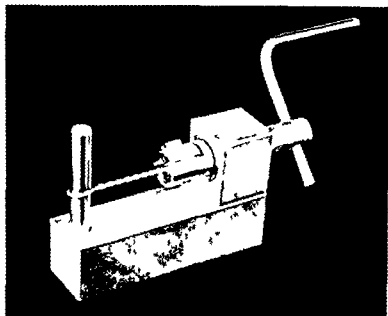


Fig. 1 Apparatus for twisting wires

Each type of wire used for the experiment was always taken from the same coil. The experiments were repeated several times with the same quality of wire from the same supplier but from different coils. The experiments referred to above were carried out on electropolished wire. 832 S1 wire with a pickled and passivated surface was found to possess the same resistance as electropolished 832 S1. A large number of coils of 832 S1 and S1 wires of the same dimensions were tested and the properties were found to be constant except on one occasion. On that occasion a distinct reduction of the resistance to corrosion was found for one coil of 1 mm S1 wire. The wires did not tolerate 3-6 turns and showed an increased tendency to corrosion at the free cut ends. Check examination of the wire by the suppliers revealed that the Mo content was too low. Preliminary investigation has shown that the minimal Mo content acceptable is 1.2-1.5 (BING & HENRIKSSON

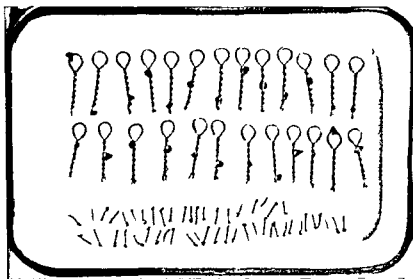


Fig 2a 832 Sh 4 turns indicator conc  $0.2\%$ .

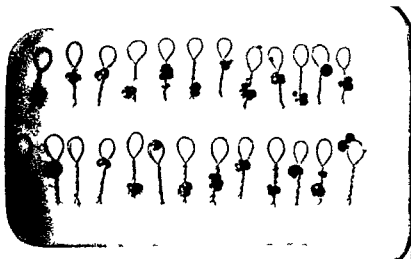


Fig 2b 832 Sh 3 turns indicator conc.  $2\%$ .

Fig 2 Twisted wires (cerclage) after exposure to ferroxyl gel for 24 hours

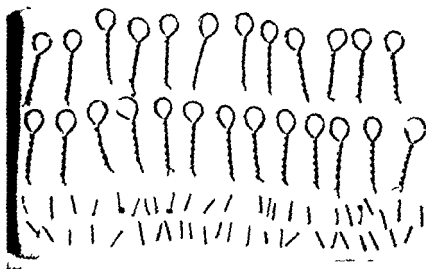


Fig. 2c. 837 SL 4 turns, indicator cone 2°.

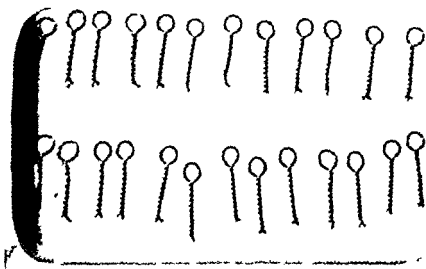


Fig. 2d. 837 SL 0 turns, indicator cone 2°.



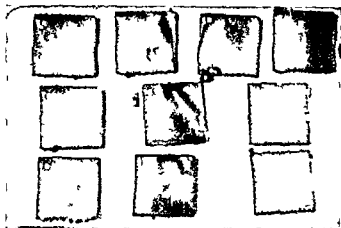


Fig. 3 Polished plates of 832 S5 after exposure to ferroxyl gel (conc of indicator 2%) for 24 hours. Note the relatively large bubbles on edges of third plate in top row and second in second row (Plates 3 and 6 in Table 2B)

(personal communication). In the case in question it was 413. This experiment clearly illustrates the value of the ferroxyl test of checking the resistance of alloys to corrosion.

### EXPERIMENTS WITH 1 MM STAINLESS STEEL SHEETS OF 3 QUALITIES

Square plates (30 mm  $\times$  30 mm) (average weight 9.2 g) were stamped out of 1 mm annealed stainless steel sheets. The cut edges were carefully ground to remove deposits and any surface damage. The surface and the edges were polished mechanically. In one corner one or two letters were stamped. A total of 60 square plates were made: 20 of 832 M, 20 of 832 S5 and 20 of 832 SL.

After the plates had been washed in soda and benzene 10 of each quality were weighed separately, after which they were placed in ferroxyl gel with 2 per cent indicator. Plates of 832 M soon showed a reaction along their edges and this reac-

TABLE 5

A

| Loss of weight K2 Mn |           |                 |           |
|----------------------|-----------|-----------------|-----------|
| First 24 hours       |           | Second 24 hours |           |
| 1 0.0012             | 6 0.0007  | 1 0.0009        | 6 0.0007  |
| 2 0.0014             | 7 0.0008  | 2 0.0008        | 7 0.0007  |
| 3 0.0017             | 8 0.0009  | 3 0.0013        | 8 0.0007  |
| 4 0.0018             | 9 0.0009  | 4 0.0012        | 9 0.0009  |
| 5 0.0016             | 10 0.0017 | 5 0.0010        | 10 0.0002 |

B

| Loss of weight K2 Mn |           |                 |           |
|----------------------|-----------|-----------------|-----------|
| First 24 hours       |           | Second 24 hours |           |
| 1 0.0004             | 6 0.0011  | 1 0.0002        | 6 0.0001  |
| 2 0.0005             | 7 0.0001  | 2 0.0002        | 7 0.0001  |
| 3 0.0018             | 8 0.0003  | 3 0.0003        | 8 0.0017  |
| 4 0.0001             | 9 0.0002  | 4 0.0002        | 9 0.0002  |
| 5 0.0001             | 10 0.0003 | 5 0.0001        | 10 0.0002 |

C

| Loss of weight K2 Mn |           |                 |           |
|----------------------|-----------|-----------------|-----------|
| First 24 hours       |           | Second 24 hours |           |
| 1 0.0002             | 6 0.0000  | 1 0.0000        | 6 0.0000  |
| 2 0.0000             | 7 0.0000  | 2 0.0000        | 7 0.0002  |
| 3 0.0000             | 8 0.0000  | 3 0.0001        | 8 0.0001  |
| 4 0.0002             | 9 0.0002  | 4 0.0000        | 9 0.0000  |
| 5 0.0000             | 10 0.0003 | 5 0.0001        | 10 0.0002 |

tion increased during the course of 1 day to assume considerable dimensions. The 832 Sk plates showed a reaction along the edges of a number of plates (see Fig. 3). 832 SL on the other hand showed only very slight edge corrosion and then only in one plate. After 24 hours the plates were removed from the gel and cleaned in soda and in benzene and weighed again. They were then re exposed to ferroxy gel for 24 hours and re weighed (Table 2).

It is clear from Fig. 3 that the losses in weight varied with the intensity of the colour. The entire 832 M series showed large blue bubbles and weight losses of 4—9 mg. 832 Sk showed such bubbles in 2 plates (Nos. 3 and 6). This is also apparent from Fig. 3.

832 SL on the other hand hardly showed any colour. A small blue patch on plate 10 might correspond to a weight loss of 0.0003. A weight loss of 0.0003 is insignificant.

It might be objected that these considerable losses of weight of 832 M and 832 Sk do not correctly reflect the rate of corrosion in 0.9 NaCl.

The ferroxy test is provocative and it is not possible to form any quantitative opinion of the rate of corrosion except by comparing the three qualities.

## EXPERIMENTAL SURFACE DAMAGE

1) In an attempt to elucidate the risks involved by stamping of letters and by unintentional surface damage liable to occur during operations or when handling appliances in a department of surgery the following experiment was performed.

2 plates  $25 \times 190$  mm with a mechanically polished surface were manufactured from 832 M and 832 Sk respectively. 30 letters (M for 832 M and Sk for 832 Sk) were stamped on each plate and underneath these letters 60 deep stabs were made in 2 rows with surgical screwdrivers of two sorts (Stille Werner no. 18987 and Stille Werner 6832—3). On exposure to ferroxy gel with 2 per cent indicator only a small attack was

TABLE 6

|  | 832 M  |  | 832 Sh   |  | 832 SL   |  |
|--|--|--|--|--|--|--|
| Type of tool used  | Number of holes with gross indication of corrosion | Number of holes with microscopic indication of corrosion | Number of holes with gross indication of corrosion | Number of holes with microscopic indication of corrosion | Number of holes with gross indication of corrosion | Number of holes with microscopic indication of corrosion |
| 2.8 mm High speed steel drill (AB Malcus)                  | 18/40  | 7/40   | 1/40   | 7/40   | 0/40   | 0/40   |
| 2.8 mm Carbon steel drill (Stille Werner 18707)            | 3/40   | 3/40   | 2/40   | 1/40   | 0/40   | 0/40   |
| 2.8 mm Vitallium drill (Austenal Inc Stille Werner 6803-4) | 15/40  | 6/40   | 3/40   | 2/40   | 0/40   | 1/40   |
| Screw driver of chromium vanadium steel                    | 0  | 0  | 0  | 0  | 0  | 0  |
| Surgical screw driver (Stille Werner 18-18087)             | 0  | 0  | 0  | 1/40   | 0  | 0  |
|  | Total 32/120                                       |  | Total 22/120                                       |  | Total 1/120  |  |

Microscopic means Magn  $\times 50$ 

seen in one M on 832 M (Fig. 4) LAING (1959) found that corrosion would occur under these circumstances

2)  $10 \times 1$  plates ( $30 \times 30$  mm) of 832 M 832 Sh and 832 SL respectively were exposed to standard effect of 3 types of drills namely 2.8 mm high speed steel drill (AB Malcus H dim stad) 2.8 mm nickel plated carbon steel drill (Stille 18707) and 2.8 mm vitallium drill (Austenal Inc Stille Werner 6803-4)

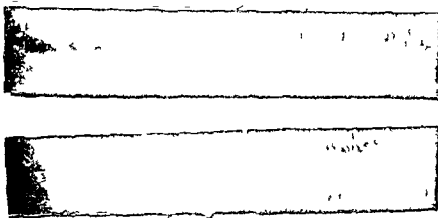


Fig 4 Letters were stamped on one plate of 832 M (upper) and one of 832 Sh. Exposure for one hour to ferroxyl gel revealed no gross corrosion in the stamped letters

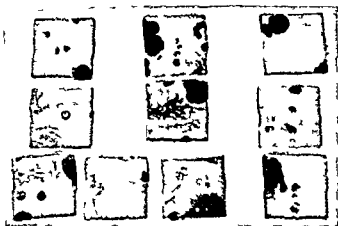


Fig 5 Ten plates of 832 M with the surface damaged by drills and screw driver scratches. The appearance of the plates is dominated by edge corrosion but corrosion is also seen in the drilled cavities



Fig. 6a 832 V with marked corrosion in several cavities

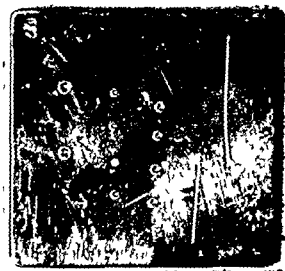


Fig. 6b 832 Sh with marked corrosion in several cavities

Fig. 6 Plates damaged by drills of high speed steel (left row) carbon steel (middle row) and titanium (right row)

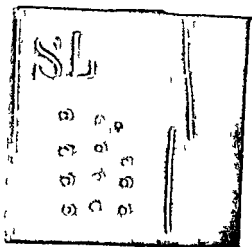


Fig 6c 832 SL with slight corrosion in one cavity

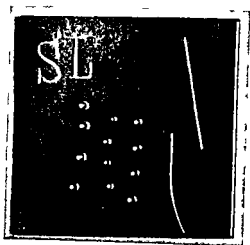


Fig 6d 832 SI without corrosion in any cavity

The drills were fastened in the usual way in a stationary electric workshop drill. The design of the experiment was such that the drill was allowed to fall against the plate from a height of 2 mm with a load of 4 kgf while the drill was rotating slowly. The drill was in contact with the plate for 2 seconds.

On each plate 4 cavities were cut with each type of drill and one scratch with a screw driver made of chromium vanadium steel and one with a surgical screwdriver (Stille Werner nr 18987). A total of 120 cavities were drilled and 20 scratches were made on each quality of steel (Types 6 a b c and d).

Steel shavings were removed by compressed air. The plates were then exposed to ferroxyl gel with 2 per cent indicator.

### Result

A reaction in some of the drill cavities appeared after a short while and was distinct after 1 hour.

The reaction first increased steadily and then ceased to increase after 24 hours.

The results were noted after 24 hours and are apparent from Table 6. Of the ten 832 M plates all showed corrosion in at least one cavity. Fig. 5. Of 832 Sk plates three showed no corrosion and of the 832 SL plates nine showed no corrosion in any of the 12 cavities. Figs 5 and 6 illustrate the experiment.

### Comments

The experiment showed that stamping of letters or figures hardly ever causes corrosion of 18/8 steel. Accidental surface damage with surgical instruments caused corrosion of ordinary 18/8 steel (832 M) when the surface damage was serious such as drilled cavities. Screw driver scratches did not appear to enhance corrosion. 832 Sk which is of the same composition as FMB steel and AISI 316 may sometimes corrode if the surface damage is deep. 832 SL usually tolerates even such deep surface damage without corrosion.



TABLL

A

| Quality 2R2   | Mo Fe | Ni Fe | Cr Fe |
|---|-------|-------|-------|
| 1   | 0 008 | 0 117 | 0 103 |
| 2   | 0 016 | 0 143 | 0 174 |
| 3   | 0 009 | 0 123 | 0 092 |
| 4   | 0 014 | 0 142 | 0 185 |
| 5   | 0 010 | 0 117 | 0 094 |
| 6   | 0 014 | 0 112 | 0 147 |
| 7   | 0 011 | 0 130 | 0 120 |
| 8   | 0 015 | 0 135 | 0 150 |
| 9   | 0 009 | 0 078 | 0 135 |
| 10  | 0 010 | 0 107 | 0 180 |
| Mixture of corrosion products<br>from several wires | 0 010 | 0 116 | 0 091 |

B

| Quality 832 S1x                                     | Mo Fe | Ni Fe | Cr Fe |
|---|-------|-------|-------|
| 1   | 0 009 | 0 169 | 0 105 |
| 2   | 0 008 | 0 168 | 0 134 |
| 3   | 0 011 | 0 197 | 0 291 |
| 4   | 0 019 | 0 232 | 0 189 |
| 5   | 0 013 | 0 180 | 0 087 |
| 6   | 0 011 | 0 166 | 0 281 |
| 7   | 0 015 | 0 164 | 0 416 |
| 8   | 0 008 | 0 161 | 0 204 |
| 9   | 0 009 | 0 072 | 0 144 |
| 10  | 0 009 | 0 147 | 0 288 |
| Mixture of corrosion products<br>from several wires | 0 012 | 0 162 | 0 186 |

### Composition of the corrosion product

As protoplasm poisons  $\text{Fe}^{+2}$ ,  $\text{Cr}^{+3}$ ,  $\text{Ni}^{+2}$  and  $\text{Mo}^{+6}$  are not equal  $\text{Fe}$  can hardly be considered severely toxic. That  $\text{Cr}^{+3}$  and  $\text{Ni}^{+2}$  are much more toxic is beyond doubt.

The artificial corrosion product obtained in the ferroxyl test of chromium nickel steel and chromium nickel molybdenum steel can be readily collected and analyzed.  $\text{Fe}$  and nickel give a deposit with  $\text{K}_2(\text{FeCN})_6$ . Chromium on the other hand gives no precipitate but a green colour which remains in solution.

The content of 10 artificial corrosion bubbles from 18-9 steel 2R2 and 10 from 17-11-2-7 steel 832 Sk was analyzed roentgen spectrographically by fillic J. Bäcklund of Westa Jernverks AB (Table 7).

Table 7 gives the X-ray intensity of  $\text{Mo}$ ,  $\text{Ni}$  and  $\text{Cr}$  in relation to  $\text{Fe}$ . Because  $\text{Fe}$  also occurs in the  $\text{FeCN}_6$  radical of the artificial corrosion product the intensity of  $\text{Mo}$ ,  $\text{Ni}$  and  $\text{Cr}$  is certainly too low compared with a real corrosion product. It would hardly be fully justified to draw conclusions about corrosion in living tissue from these analyses of the artificial corrosion products in the ferroxyl test. In the ferroxyl test the products of corrosion contain nickel in roughly the same proportion as does the original alloy.

### EXPERIMENT WITH CREVICES (1)

In all surgical cases where screws and plates, screws and flanges or nuts and bolts are used crevices may occur. Technically crevices imply a risk of stagnation of electrolytes and concentration of electrolytic products present. Further a reduction of the oxygen concentration may occur in the crevice. This probably holds also for metal appliances deposited in human living tissue.

A few preliminary experiments were performed first. The 10 plates of 838 M, 832 Sk and 832 SL each which were used for studying surface damage caused by drill and chisel

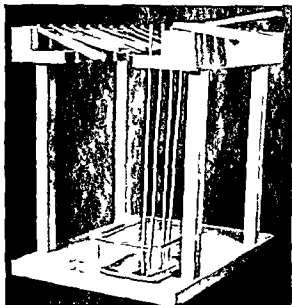


Fig. 7 Frame used for the experiments with crevices between ends of rods and surface of plates

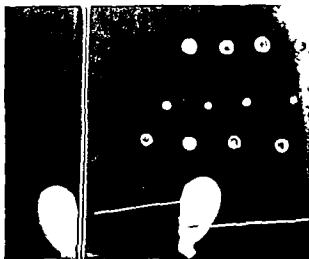


Fig. 8 Corrosive areas between superimposed plates of 83% Ni

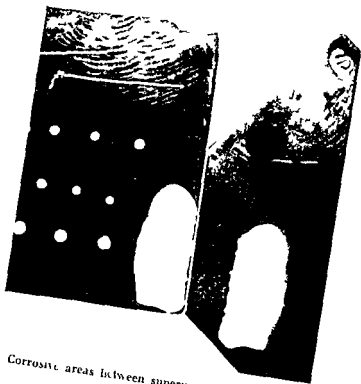


Fig 9 Corrosive areas between superimposed plates of 832 Sh

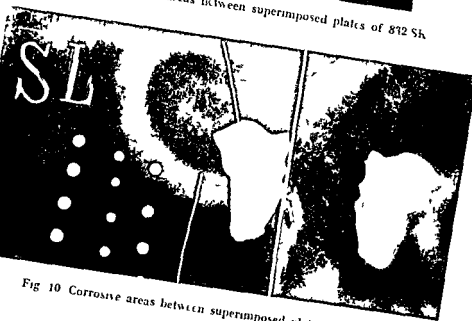


Fig 10 Corrosive areas between superimposed plates of 832 SL

scratches were placed in the liquid gel (0.9 % NaCl and 2 % indicator) one on the other so that the gel could fill the crevices before the next plate was deposited. Undamaged surfaces were placed against damaged surfaces. In addition the plates were placed like roof tiles over one another.

After a day (24 hours) Turnbull blue was seen to ooze from the crevices of 832 M but not from those of Sk and SL. The experiment was continued for a week. Then abundant corrosion bubbles were seen in several crevices of 832 M and in crevices of 832 Sk but not of 832 SL.

Plates of 832 M were held together firmly by Turnbull blue. This also applied though to a less extent to two plates of 832 Sk and SL. Between 10 plates there are 9 crevices. 832 M showed deep corrosion in 3 of the crevices (Fig. 8). 832 Sk showed a deep area of corrosion between plates 6 and 7 (Fig. 9). 832 SL likewise showed a large but superficial area of corrosion between plates 4 and 5 (Fig. 10). The corrosion areas were mirror pictures of one another as is apparent from the illustrations.

Exactly the same experiments were carried out on 10 plates with intact surfaces of polished plates of 832 Sk and of SL. This time the experiment was continued for 2 days. Then corrosion areas were seen in 4 crevices of 9 in 832 Sk and in one of 9 in 832 SL. In the last case the corrosion was situated between the third plate and the fourth.

### Comments

In this experiment the ferroxyl test was used not to demonstrate corrosion by colour indication but provoke corrosion. It soon produced  $\text{Fe}^{2+}$  and readily demonstrable damages. Such severe damage as that observed in the latter experiment was not seen in any of the other experiments with ferroxyl test on polished surfaces of chromium nickel or chromium nickel molybdenum steel. This might be an illustration of crevice corrosion.



Fig. 11

## EXPERIMENT WITH CREVICES (2)

When fractures are fixed with plates and screws there will always be crevices between the head of the screw and the plate (see Fig. 11)

A method for studying crevices of this type was devised. In a frame of the type shown in Fig. 7 20 rods can be placed against plates of the same alloy. The plates are placed in a vessel containing ferroxyl gel (2% indicator). The rods can be loaded via a lever and be pressed against the plates with any desired moderate pressure.

A series of experiments were performed to compare the three alloys 832 M, 832 SL and 832 SL (see Table 2). The alloys were tested as rods 4 mm in diameter and 300 mm long with a weight of 28 gram. These rods were made of uncoiled wire from the same coil and were thus probably of the same batch. One end of the rod was ground flat and the other ground to a fine point. The ends were then surface treated by pickling and passivation in  $\text{HNO}_3$ .

## Results

The results are apparent from Figs. 12—14.

832 M 1 A Tip against plate produces corrosion at two sites. (The corrosion at site of rod 4 is due to edge corrosion and is not located at the tip of the rod) Fig. 12 a

1 B Ground plane end against plate produces strong corrosion at 9 sites Fig. 12 b

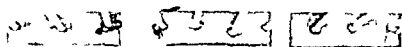


Fig 12 a Pointed rods resting on plates as seen from above. Rods and plates of 83° Al. Note edge corrosion in the middle plate

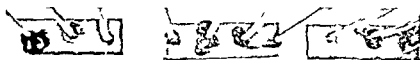


Fig 12 b As in fig 12 a but ends of rods ground plane

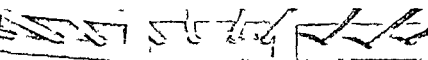


Fig 13 a Pointed rods resting on plates. Rods and plates of 83° Sb

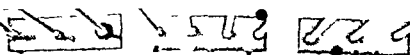


Fig 13 b As in fig 13 a but ends of rods ground plane

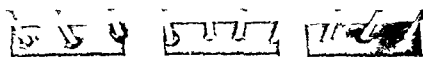


Fig 13 c As in fig 13 b but rods loaded with 500 g

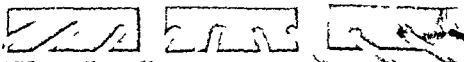


Fig 14 a Pointed rods resting on plates. Rods and plates of 832 SL

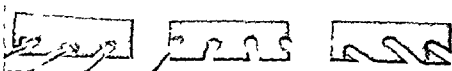


Fig 14 b As in fig 14 a, but ends of rods ground plane

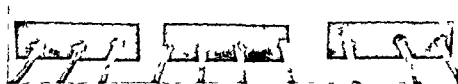


Fig 14 c As in 14 b but rods loaded with 500 g

- 832 Sk 2 A Tip against plate causes no corrosion Fig 13 a  
 2 B Ground plane end against plate causes strong corrosion at site of one contact. Several sites of corrosion along the edge of the plates Fig 13 b
- 832 SL 3 A Pointed end against plate causes no corrosion Fig 14 a  
 3 B Ground plane end against plate cause no corrosion Fig 14 b

After the rods had been reground and resharpened they were loaded with 500 g and thus pressed against a new series of plates<sup>1</sup> with a 20 fold force. The rods weighed about 28 g

<sup>1</sup> All plates in each quality were made of the same sheet



- 832 SK 2 C Pointed end against plate causes no corrosion  
 2 D Ground plane end against plate causes severe corrosion at 3 points of contact Fig. 13 c
- 832 SL 3 C Pointed end against plate causes no corrosion  
 3 D Ground plane end against plate causes severe corrosion at one point of contact See Fig. 14 c

All photographs were taken after the test had been allowed to proceed for 12 hours but incipient corrosion could be detected within 5—10 minutes

After 1 hour it was always possible to predict what the results would be after 12 hours Figs. 15—17 illustrate corrosive areas after experiment 1 A and 1 B

When a metal plate is fixed by screws the surfaces of the plates and the screws are pressed against one another with considerable force. The design of the present experiments was not such as to ascertain whether this pressure favours corrosion or whether corrosion is due entirely to the crevice between the surfaces. Spot contact with the tip of the rods however appeared to favour corrosion less than contact between two plane surfaces. The sharp points of the rods loaded with 500 g produced no signs of corrosion of 832 SK or SL. Judging from experiments with loaded plane bottomed rods the interspace between the bottom of the rod and the supporting surface will possibly decrease with increasing load of the rod and it is known that the risk of corrosion increases as the crevice becomes very small.

No attempts were made to ascertain the effect of the last turn or so when a screw is screwed tight on the surface of the threading and head of the screw or on the surface of the plate through which it is screwed.

### Comments

It is clear that 832 M was unable to satisfy the requirements placed upon it by the experimental conditions.

832 SK was evidently sensitive to small crevices and like 832 M failed to stand the present test.



Fig 15



Fig 16

Fig 15 Corrosive areas on plane ends of rods of 832 M after exposure to ferroxyl gel in the way seen in fig 12 b ( $\times 5$ )

Fig 16 Corrosive areas in plate of 832 M on which rods in fig 15 had been placed



Fig 17 Corrosive areas on jointed ends of rods of 832 M after exposure to ferroxyl gel in the way shown in fig. 17a ( $\times 5$ )

832 SL was superior to the other two alloys but crevice corrosion was nevertheless noted in one sample

Although it is too early to say in what measure the present experimental findings apply to plates sunk and screwed in human bone and with due reservation for the provocative nature of the ferroxyl test it would appear that 832 SL best fills the requirements for orthopaedic surgery

#### EXPERIMENTS WITH CONTACT BETWEEN DIFFERENT ALLOYS AND ELEMENTS

Handbooks on corrosion are liable to give the less critical reader the impression that so called stainless steel alloys can be used in combination with vitallium for example in metal



Fig 1a



Fig 1b

Fig 1a Corrosive areas on plane ends of rods of 832 M after exposure to ferroxyl gel in the way seen in fig 12b (< a)

Fig 1b Corrosive areas in plate of 832 M on which rods in fig 1a had been placed

TABLE 8

*Galvanic series for different metals and alloys**Electrode potential measured in saltwater against saturated calomel electrode ranging from  $-1.1$  for magnesium alloys to  $+0.33$  for platinum*

|  |   |
|--|---|
| 1 Magnesium alloys                               | 25 Inconel (80 % Ni 20 % Fe 10 % Cr) active |
| 2 Zinc   | 26 Titanium active                          |
| 3 Galvanized steel and iron                      | 2 80—90 Ni Cr alloy                         |
| 4 Aluminium pure and Al with out copper          | 28 Brass                                    |
| 5 Aluminium of compound type with anodic coating | 29 Admiralty brass                          |
| 6 Cadmium alloys                                 | 30 Aluminium brass                          |
| 7 Aluminium with copper                          | 31 Tombac                                   |
| 8 Carbon steel                                   | 32 Nickel silver                            |
| 9 Steel with an addition of copper               | 33 Copper                                   |
| 10 Cast iron                                     | 34 Copper nickel alloy                      |
| 11 Chromium steel 4—6 % Cr active                | 35 Siliceous bronze                         |
| 12 Chromium steel 12—30 % Cr active              | 36 Monel metal                              |
| 13 Ni alloyed cast iron                          | 3 Silver solder                             |
| 14 18—8 stainless steel active                   | 38 Nickel passive                           |
| 15 18—8 stainless steel with 3 % Mo active       | 39 Inconel passive                          |
| 16 Tinman's solder (lead + tin)                  | 40 80—20 Ni Cr alloy passive                |
| 17 Lead  | 41 Chromium steel (19—18 % Cr) passive      |
| 18 Tin   | 42 18—8 stainless steel passive             |
| 19 Muntz metal                                   | 43 Chromium steel (23—30 % Cr) passive      |
| 20 Manganese bronze                              | 44 18—8 stainless steel with 3 % Mo passive |
| 21 Tolran bronze                                 | 45 Titanium                                 |
| 22 Marine bronze                                 | 46 Graphite                                 |
| 23 Phosphor bronze                               | 4 Amalgam                                   |
| 24 Nickel active                                 | 48 Quicksilver                              |
|  | 49 Gold                                     |
|  | 50 Titanium passive                         |
|  | 51 Platinum                                 |

In combinations with direct contact of stainless steel and titanium the steel will be the anode. This is what also happens when stainless steel and titanium are in direct contact.

On unintentional combination of titanium and titanium

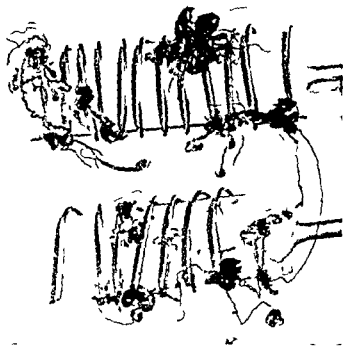


FIG. 18 8.2 M wire wound round titanium plate and exposed to ferroxyl gel for 24 hours. Observe massive corrosion.

titanium will be the anode and titanium will lose substance if the passive layer is not intact. It is not likely that titanium can be activated in 1 % chloride environments.

### Experiments

1. Electropolished 1 mm wire of 2R2 was wound round titanium plates 12 times round one plate and 6 times round the other. There were thus a total of 48 and 24 points of contact respectively. The plates were then placed in ferroxyl gel (2 % indicator) and after 3-4 minutes Turnbull blue began to ooze from a number of the points of contact between the wire and the plate. After 1 hour Turnbull blue was abundant (Fig. 18).



Fig. 19 832 Sh wire in contact round vitallium plate and exposed to ferroxyl gel for 24 hours  $Magn \times 10$  Note red slight corrosion

2 The experiment was repeated with the same vitallium plates and 1 mm electropolished 832 Sh. In the beginning no sign of any reaction could be demonstrated but after 1 hour small spots of blue could be recognized with the naked eye. On examination under the microscope a blue colour was seen at several of the points of contact (see Fig. 19). After 1 day faint but gross blue spots were seen. Under the microscope such spots were seen at all points of contact (Fig. 19). The experiment was extended to include cutting off the wire with pliers under the surface of the gel and then signs of corrosion soon appeared at the cut surface. These ends became intensely blue within 1 hour.

3 The experiment was repeated with 832 Si. No macroscopic or microscopic signs of corrosion appeared within 1 day (Fig. 20). When the wire was cut in lengths as in previous experiment marked corrosion occurred within 1 hour.

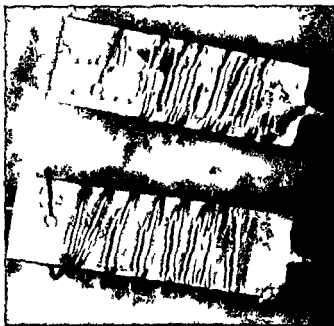


Fig. 20 832 S1 wire wound round titanium plates and exposed to ferroxyl gel for 24 hours. No corrosion indicated

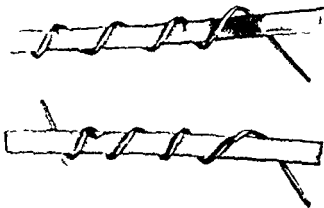


Fig. 21 832 S1 wire wound round rectangular titanium rods. Corrosion only slight



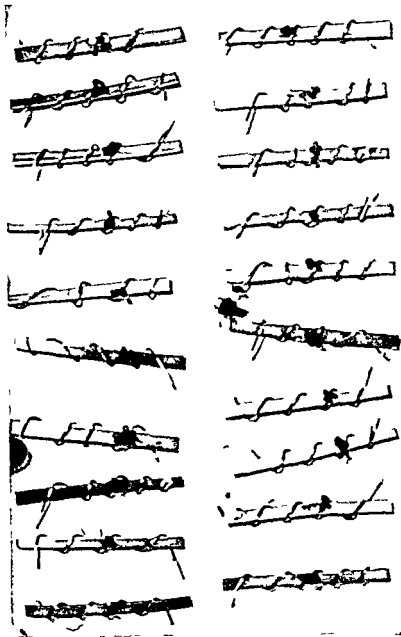


Fig 22 83° Sk wound round rectangular titanium rods. The wire was divided roughly in its middle without taking it out of the gel. Note constant gross corrosion in the cut ends. This picture was taken 2 hours after the wire was divided.

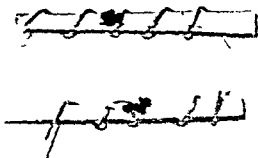


Fig. 23 Enlargement of first two samples illustrated in fig. 22

4. 1 mm polished 2R2 was wound 4 turns around 20 fil 1 mm square rods thus 16 points of contact between each rod and the wire with a minimum of 320 points of contact between the wire and all the rods. The rods were then deposited in ferroxyl gel. After 30 minutes many sites of contact showed signs of corrosion. After 1 day a large number of the sites of contact showed corrosion. Only one of the 20 samples showed no such corrosion.

5. Same experiment as in 4 but now with 832 Sk. After 30 minutes several points of contact showed corrosion. On examination under the microscope practically all points of contact showed corrosion (Fig. 21). When the wires were cut under the surface of the gel all the cut surfaces showed corrosion within 2 hours (Figs 22 and 23).

Exactly the same experiment was performed with 832 SL but now the results were different. No corrosion was seen within 30 minutes. After 24 hours gross corrosion was seen at one site and microscopic corrosion at 4. When the wires were cut under the surface of the gel all showed corrosion within 2 hours — almost the same picture as in the experiment with 832 Sk (Fig. 22).



FIG 24 Corrosion in a vitallium plate around which titanium wire had been wound the corrosion was due to a flaw in the plate Magn  $\times 70$

### Comments

These experiments which can only be regarded as preliminary but they nevertheless show that under some experimental conditions annealed wire of 832 SL quality is so passive that if it is absolutely intact it does not corrode on direct contact with vitallium and titanium. The slightest damage such as on cold working can however activate even this steel with galvanic corrosion as a result.

Steel of type 832 SL might occasionally retain its passiveness but even the slight cold working, e.g. cutting with pliers or crevices can occur and activate the steel.

### WHAT ARE THE RISKS OF UNINTENTIONAL USE OF VITALLIUM AND TITANIUM IN COMBINATION?

According to the standard electrode potential series vitallium will be the anode and titanium the cathode. Thus vitallium should be able to undergo corrosion if its passive surface is broken and the vitallium is active.

A number of experiments have been made with direct contact between titanium and vitallium. Series of vitallium screws

(Austenil Inc.) were inserted in wooden blocks and titanium wire was wound tightly around the screws. Despite the numerous crevices between the turns of the wire and the screws no corrosion of vitallium was demonstrable within a week.

Similar experiments were carried out with titanium wire wound around vitallium plates. The surface of one plate had been damaged by drilling 120 cavities. On one occasion one small spot of corrosion was demonstrated after 3 days in one vitallium plate (Fig. 24). The corrosion was seen at a site of a flaw in the plate but not in any drill cavity. The corrosion product was analysed x-ray spectrographically (Backlund) and was found to contain Co, Cr and Mo.

### Comments

Judging from the experiments it seems that hard vitallium has a very passive surface and that even on contact with titanium it corrodes only exceptionally. LARV *et al* suggested that there might be a risk of corrosion of vitallium screws screwed with steel screw drivers owing to transfer of metal from the latter to the screws. Judging from experiences with the strong passiveness of hard vitallium even on contact with titanium it may be questioned whether there really is any risk of corrosion of vitallium under these circumstances.

If the tools are made of steel any particles torn from them will of course go into solution but since they will be anode in relation to a vitallium screw for example and be cause of their smallness they will be rapidly dissolved (Fig. 28). Why the cathodically protected vitallium should corrode under such circumstances when it tolerates a cleft in contact with titanium is difficult to understand.

PETERSEN & LARSEN (1960) pointed out that corrosion (local action) could not be produced experimentally with hard vitallium made by Austenil Inc.

On one occasion hard vitallium in direct contact with titanium showed signs of corrosion in the ferroxyl gel test (see above) (Fig. 24).



Fig 25 Plate made of cobalt alloy of approx the same composition as vitalium and placed in ferroxyl gel (2% indicator) without any previous treatment whatsoever Note slight corrosion  $Magn \times 20$



Fig 26 Screw of same alloy as plate in fig 25 Note corrosion though screw was brand new placed in ferroxyl gel with 2% indicator  $Magn \times 20$

It should be observed that the manufacture of vitalium is a complicated process (as pointed out by VENABLE in 1948) Not all manufactures can produce a really homogeneous material Austenal Inc however appear to have overcome these difficulties

Attempts have been made in Sweden to manufacture Co alloys of stellite type resembling vitalium and specimens have been sent to the department of Med Physics in Lund for testing for medical use

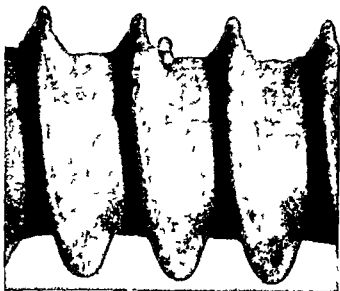


Fig 27 As in fig 26 but ferroxyl gel contained only 0.2% indicator  
Magn  $\times 20$



Fig 28 Vitallium screw screwed in wooden block with driver of chromium vanadium steel After exposure to ferroxyl gel the transfer is demonstrable within a few minutes (Dark colour in the picture)

The plates were measured for hardness and studied for corrosion in the ferroxyI gel test. The hardness varied from 17 to 34 Rc (Rockwell C).

In the ferroxyI test punctate corrosion occurred within 24 hours (Figs 25, 26 and 27).

The variation in the hardness was sufficient to suspect structural differences in one and the same plate so that local elements could arise. This was also confirmed by the ferroxyI test.

During the last months an improved domestic stellite has been tested at the Department of Med. Physics. It appears that this quality is less corrosive than the quality tested in the present investigation.

It may thus be concluded that reliable stellite plates can only be cast by highly specialized manufacturers.

## SUMMARY

The risks of corrosion under some conditions resembling those of surgery have been studied with the aid of the ferroxy test. Under the experimental conditions it appears that

- 1 Cold working (cold-chamber) of
  - a) 2R2 and 832 Sk involves a serious risk of corrosion
  - b) 832 SL will tolerate twisting but less well cutting with pliers
- 2
  - a) Slight surface damage (screw driver scratches) and stamping of letters involves no risk in either quality
  - b) Deep surfaces damage (drill cavities) involves a serious risk in 832 M, a moderate risk in 832 Sk and practically no risk in 832 SL
- 3 Crevices involve
  - a) a serious risk in 832 M
  - b) a moderate risk in 832 Sk
  - c) a slight risk in 832 SL
- 4 Contact with vitallium involves a
  - a) serious risk of gross corrosion in 2R2
  - b) serious risk of minute corrosion in 832 Sk
  - c) serious risk of gross corrosion in 832 Sk and 832 SL if the steel is severely cold worked as it sometimes is in bone surgery
- 5 Contact with titanium involves the same risks for the three steel qualities as those stated under 4 (vitallium)
- 6 Vitallium ought to stand contact with titanium except under very unfavourable conditions

It appears that chromium-nickel-molybdenum steel with a content of 17.5 % Cr, 14 % Ni and 4.5 % Mo is superior to steel with 17.5 % Cr, 11 % Ni and 2.7 % Mo under the experimental conditions used.



## CLINICAL CONSIDERATIONS

It is more difficult to assess the chemical effect of plates screws pins and wires in the human body than to judge the effect of metal implants in experimental animals or in vitro. In the first place it is necessary to rule out all effects of surgical technical procedures and infection.

When this is done the mechanical effect of a foreign body must be considered.

Comparison of the degree of chemical effect with that of mechanical requires detailed records.

When large appliances are used it would appear that the mechanical component of the cause of the reaction is greater than the chemical. A Huentscher nail in the femur must of course imply a severe mechanical effect just as will a MacLaughlin nail with a plate.

General clinical experience has however shown that the benefit obtained by these applications is so considerable compared with conservative methods that they more than outweigh the risk of infection and mechanical or chemical irritation.

Since the mechanical factor has been accepted as unavoidable in bone and joint surgery the chemical component has come to the foreground in recent years although it may be a factor of less importance.

In the 1930ies and 1940ies the chemical component was brought under fair control by the introduction of high alloyed chromium nickel steel of so called 18 8 type (LANCE 1926 JONES & LILBERMAN 1936 HUDOCK 1940 SHERMAN 1940 KEY 1941). The still better qualities of chromium nickel molybdenum steel were shown by SHERMAN (1940) and KEY (1941).

The stellites particularly vitallium were introduced by

VLAHLE & STUCK (1937-1947) Despite attempts in USA (1947) and in Great Britain (1956) to limit the selection of types of steel for osteosynthetic purposes to chromium nickel molybdenum steel (AISI 316 and FMB) it has been found that even this type of steel shows corrosion spots after it has been in the body for some time (ZATFE 1955 SCALES 1955 SCALES & ZARLEK 1955 HICKS 1958 SCALES *et al* 1959 COHEN *et al* 1959)

Vitallium is so inert that SCALES & ZARLEK (1955) and SCALES *et al* 1959 concluded that they never found any corrosion spots in any vitallium implants that had been in the human body

At the Department of Orthopaedic Surgery in Lund all cases in which surgical implants have been removed are receiving special attention. Special notes are made of the clinical course, histological findings of biopsy specimens and the results of physical and chemical investigations of the extracted implants which includes

- 1 Gross inspection
- 2 Examination with magnet
- 3 Roentgen spectrographic analysis
- 4 Microscopical examination
- 5 Corrosion test (ferroxyl test)

A clinical investigation as mentioned above does not yield such reliable information as do experimental studies because it is hardly possible to judge the reaction of the tissue around the entire nail or plate except of course at autopsy. Investigation must be limited to examination of a piece of tissue from the region where the surgeon has found the tissue reaction to be most intense. In addition in a clinical investigation it is difficult to assess how much of the tissue reaction is due to mechanical irritation and how much to chemical

Since many sorts of alloys are used, any collection must be relatively large to permit comparisons. The material hitherto collected at the department is not yet large enough to permit comparisons with any degree of certainty. The results will be published later.

On one point, however, the data obtained are of special in-

interest namely indications of corrosion by the ferroxyl test. The information obtainable by this test will be apparent from a few examples given below.

## INTERPRETATION OF FERROXYL TEST PERFORMED ON EXTRACTED SURGICAL IMPLANTS

It is known from metallurgical experience that chromium nickel molybdenum steel is passive in 0.9 per cent sodium chloride at a temperature of about 40° C provided it is not exposed to additional corrosion promoting factors. A Rush pin approaches the ideal in the absence of supervening corrosion promoting factors see Figs 29.

A Sven Johansson nail possesses similar though not quite so satisfactory properties. It is however subjected to somewhat greater stress and cold working on insertion<sup>1</sup> and also to metallic transfer from the guide wire in the central canal see Figs 30 a and b.

A Kuentscher nail has the same properties as the Sven Johansson nail but on insertion it is subjected to a fair degree of cold working, and particularly the U shaped femoral nail can be damaged quite a lot by the guiding pin. Figs 31 and 32.

The classical plate with screws is exposed to three corrosion promoting factors: severe cold working of the screw heads, transfer of metal from the screw driver to the screw and last but probably not least the crevice between the plate and the screw heads see Figs 33 a b c and d.

Attention has been drawn to the transfer of metal from tools to plates and screws by LAING *et al* (1955, 1958 and 1959) Figs 28 and 30. In the present investigation the ferroxyl test was also used to study the transfer of metal from the guide in the central canal of three and four winged collum nails made of vitallium. As previously mentioned if only

<sup>1</sup> It is obvious that removal of a surgical appliance can damage it. This might cause corrosion as well as damage on insertion and must be born in mind when judging the ferroxyl test after extraction of an appliance.

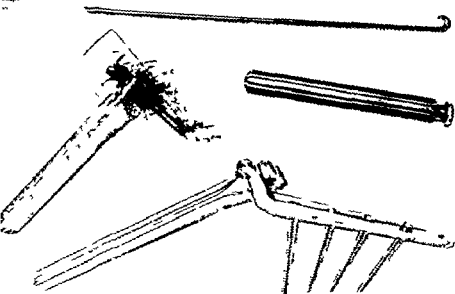


Fig. 3 Ferroxyl test (indicator concentration 2 per cent) of Sven Johansson nail Mc Laughlin nail and plate Aronson nail and Rush pin. Photo graph taken 2 hours after beginning of test. Only the Aronson nail shows corrosion.

*Sven Johansson nail.* Analysis: Cr 17.5%, Ni 14% and Mo 4.5% (Vesta 832 St). Clinical: The nail was extracted two weeks after insertion because it had projected through the femoral head. The nail was removed with an extractor of conventional type.

*Mc Laughlin nail* of titanium without signs of corrosion or transfer of metal from the tool to the nail. Clinical: Nail and plate removed three months after insertion because the fracture had healed but the nail threatened to project through the cranial part of the femoral head. Extraction revealed a large abscess under fascia lata. There were no clinical signs of infection and the surgeon was surprised by the finding. The entire plate was covered with oedematous purulent granulation tissue. Culture of the discharge gave no growth. Pathologist's report: oedematous granulation tissue with abundant Turnbull positive pigment.

*Aronson nail.* Analysis: Cr 25%, Ni 5% and Mo 1.5%. Clinical: Nail removed 4 years after insertion after the fracture had healed because patient had moderate tenderness and pain over the head of the nail. The nail was difficult to remove because the screw was tightly fixed in the canal through the nail. Removal of the screw thus implied a considerable amount of cold working on the screw head. Ferroxyl test showed severe



Fig 30a + b Detailed view of head of Swen Johansson nail in Fig 29 (X) 24 hours after beginning of test. The black dots on the nail are in reality blue. They might be due to transfer of metal from the extractor and possibly persistent from transfer on insertion.

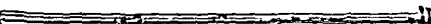


Fig 31 Ferroxyl test of Kuentscher's nail in glass tube. Indicator concentration 2%. Photograph taken 7 hours after beginning of test. Nail analysis: 18% Cr, 8% Ni and 1.5% Mo rest Fe. Clinical Nail removed 7 months after insertion when femoral fracture in 18 year old female had healed. Corrosion at base in extraction eye along the bridge of the nail and the edges of the U channel as well as severe corrosion in the inside of the U channel.

corrosion of the screw head (effect of cold working) and of the canal of the screw (crevice corrosion).

**Rush pin.** Analysis: Cr 18%, Ni 10%, and Mo 2%. Clinical: The nail was removed three months after insertion after fracture had healed. It was removed for prophylactic reasons. No indication of corrosion.



Fig. 3: Detail ( $\times 10$ ) of a part of Fig. 31 where corrosion was strongest

Cause of corrosion 1) Unsatisfactory composition of the alloy

2) Cold working of nail on insertion

3) Transfer of metal from guide to nail



Fig. 3a: Blount's plate in ferroxyl gel. Indicator concentration  $2 \times 10^{-3}$  M. Graph taken 7 hours after beginning of test. Analysis not performed. author Zimmer 3160 stainless steel which is usually to be understood as 18% Cr 8-11% Ni 2-3% Mo. Clinical plate removed 3 months after insertion as internal fixation of Mc Murray osteotomy after collar pseudarthrosis. Corrosion along edge of plate where the surface is severely damaged in several places. Entire plate covered by small surface scratches without corrosion. Abundant corrosion of screw hole. Note the nail blade shows no corrosion.

moderate or slight such deposits will soon be dissolved by the body fluid especially if the nail has been in living tissue for a long time. This is supported by the fact that the central canal test is often negative. In those cases where the test is positive one might imagine that the transfer would



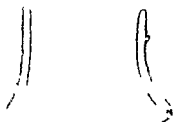
Fig. 33b This screw head ( $\times 3$ ) was deformed by the screw driver but shows hardly any corrosion. The screw hole however shows corrosion. The colours seen to ooze from underneath the plate around the screw come from the screw hole. This may be an example of crevice corrosion. In one of the larger areas damaged on the edge of the plate some corrosion is seen. No corrosion is seen in the stamped letters and figures.



Fig. 33c Detail of middle screw ( $\times 3$ ). Edges of plates severely damaged and corroded. Screw head shows hardly any corrosion. Abundant corrosion in screw hole.



Fig. 33d Detail of uppermost screw in plate ( $\times 3$ ). Severe surface damage on edge with corrosion. Screw head markedly corroded. This may be an example of the total effect of the transfer of metal from a tool to the screw and of cold working.



central canal test from collum nail made of vitalium  
 and with a wooden ear pin. The diameter of the  
 slightly less than that of the canal. Gel was poured  
 after it had set it was pressed out with a similar ear  
 pin. If the test is positive or only weakly positive it can be pressed out  
 as a worm. It will of course be somewhat compressed and  
 the nail. Any persistent gel can be readily expelled.  
 If the wooden pin is then rolled over white filter  
 pieces of Turnbull blue will become apparent.

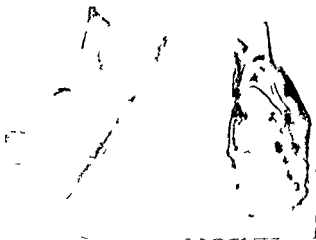


Fig. 141 Positive central canal test ( $\times 10$ ). If the test as in this case  
 is positive the gel will not always be expelled as a single worm but in  
 fragments due to the gel adhering to the wall of the central canal where





Fig 34c The strongly positive central canal test from a collum nail of vitallium ( $\times 10$ ). The dark coloured gel attached to the ear pin is intensely blue. This large amount of Turnbull blue probably indicates transfer of metal from the guide that had been used. In this case it was difficult to pass through the earpin to clean the canal before the test.

that part of the transfer had persisted as rust in the canal. This deposit may possibly be one of the main sources of the iron pigment seen in granulation tissue around the heads of femoral nails made of vitallium (EMMETS 1957).

A negative, a slightly positive and a strongly positive central canal test are illustrated in Figs 34a, b and c.

If two different metals or alloys are in contact with crevices as e.g. plate screw there is great risk of corrosion of the metal which is less noble (i.e. anode in the element).

This gives rise to a genuine galvanic corrosion.

the small deposits are located. The dark coloured patches in the dry gel to the right may represent metal transferred from the guide. It is possible though less likely that they may be due to rests from the manufacturing of the nail or iron in the vitallium. A further possibility is that it might be iron from decomposed haemoglobin. It can be broken down to a sized form. An accumulation of blood could be sealed in the central canal and there broken down to inorganic form but this sounds less likely.



Fig. 3a. Detail of Wright's plate of vitallium from angle between the nail blade and the screw blade ( $\times 20$ ). The photo was taken 24 hours after the beginning of the test. Clinical. Before insertion of the plate the latter had been heated and the angle increased from 80 to 130 degrees. The plate had been bent at that place represented by the picture. The plate had been used to fix a McMurray osteotomy. The plate was removed 8 weeks later because of dislocation of the osteotomy. The ferroxyl test indicates corrosion at the site where the plate had been bent. The bubbles are dark brown. Roentgen spectrographic analysis of the artificial corrosion products showed Co, Cr and Mo with predominance of Co.

Transfer of tool steel to the vitallium will likewise give rise to such galvanic element. In such cases rapid dissolution of the steel alone may be expected (Fig. 28).

Stellites (Co-Cr) are present in vitallium and neutrillium. Vitallium is very resistant to corrosion. VENABLE & STUCK claimed this alloy is superior as early as 1937 and proved it in 1947. The reason why this has not generally been accepted might be explained in the following way. A Rush pin made of chromium nickel molybdenum steel is almost as good as vitallium as far as corrosion is concerned. This may perhaps also

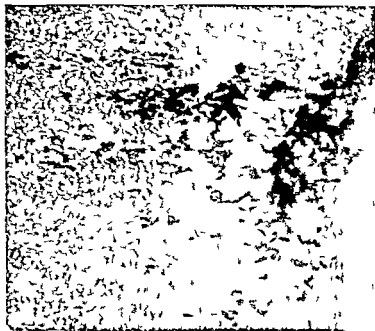


Fig. 6 Detail of plate adjacent to the angle of the same plate as in fig. 3a ( $\times 20$ ). In order to bend the plate to desired angle the plate might have been gripped with a pair of tongs in this place. The dark coloured areas in the photograph are in reality intense blue. The iron demonstrated probably represents material transferred from these tools.

hold for a Sven Johansson nail. In addition other properties of this steel may be preferable compared with vitallium.

However in association with cold working and crevices in appliances such as a Blount's plate (Fig. 33) or Aronson nail (Fig. 29) the weak point of the steel with respect to corrosion will become apparent. In spite of the fact that LAING (1959) for example pointed out that vitallium might also be susceptible to crevice corrosion clinical experience (ZAREK & SCALES 1955, SCALFS 1956 and SCALES *et al.* 1959) suggests that in this very respect it is superior. In large appliances consisting of a number of details stellites appear to be superior to chromium nickel molybdenum steel as far as corrosion is concerned.

On one occasion the ferroxyl test showed corrosion of a titanium plate made of Austenit 1185 35 and 36. In that case the surgeon had heated the plate and bent it for a special purpose. It is obvious that the manufacturer's guarantee cannot hold for material worked in this way. This example is only given to show that the ferroxyl test will also show corrosion of cobalt alloys.

The so called microcorrosion of titanium and pure titanium made probable by FERGUSON, LAINE & HODGE 1960, LAMNUS & STENRAM 1960 and LAMNUS, STENRAM & BAICKELUND 1960 is still obscure and its clinical significance cannot be judged.

## References

- BECHTOL C O FERGLSON A B JR & LAING P G Metals and engineering in bone and joint surgery The Williams & Wilkins Co Baltimore 1955
- BERG S Personal communication
- BERG S & HENRIKSON S Personal communication
- BLUNT J W JR HUDACK S S & MURRAY C R Clin Congr Am Coll Surg New York Sept 1959 Cit Metals and engineering in bone and joint surgery The Williams & Wilkins Co Baltimore 1959
- BOWDEN F P WILLIAMSON J B P LAING P G J Bone Jt Surg 3 B 66 1955
- BOTHF R T BEATON L E & DAVENPORT H A Surg Gyn and Obst 41 598 1940
- COHEN J HAMMOND G J Bone Jt Surg 41 A 524 1959
- CLARKE L G C & HICKMAN J J Bone Jt Surg 37 B 461 1953
- CLARKE L G C HICKMAN J & JENNINGS A R J Bone Jt Surg 40 B 99 1958
- LMNÉUS H Acta Orthop Scand 6 736 1956/57
- Acta Orthop Scand Suppl 42 A 1960
- LMNÉUS H & PETERSEN P Nord Med 59 593 1958
- Acta Orthop Scand 9 fasc 4 431 1960
- LMNÉUS H & STENRAM L Acta Orthop Scand 29 fasc 4 315 1960
- LMNÉUS H STENRAM L & BAECKLUND J Acta Orthop Scand 30 276 1960
- FERGLSON A B JR LAING P G & HODGE E S J Bone Jt Surg 42 A 1960
- FINK C G & SMITH J S J Electrochem Soc 91 71 1948
- HENSCHEN C & GERLACH W Zbl f Chir 61 828 1934
- HICKS J H In Modern Trends in Surgical Materials Ed. Gillis London 1958
- HUDACK S S Arch Surg 40 867 1940
- JERGENSEN F H Rem Cru Titanium Rev 7 6 1955 Cit Down Bros and Mayer & Helph Ltd 1960
- JONES I & LIEBERMAN B A Arch Surg 32 990 1936
- JÜRGENSEN H J Experimentelle Undersøgelser over Fracturhelings ved Osteosyntes Disp Copenhagen 1941

- HALLING B & LILJEKVIST G Cellulosa och I upper I d Svenska Pappers  
och Cellulosaingenjorsforeningen Stockholm 1948
- KEY J A Arch Surg 43 615 1911
- Surg Gynec and Obstetr 82 313 1946
- KUTZFELNIG A Werkstoffe und Korrosion 3 16 1960
- LANGE M Ztschr f Orthop Chir 47 590 1906
- LEVINTHAL G S J Bone Jt Surg 33 A 473 1951
- Amer J Surg 34 135 1951
- LAING P G J Bone Jt Surg 40 A 853 1958
- In Metals and engineering in bone and joint surgery The Williams  
& Wilkins Co 1953
- LAING P G MEDANCA L R & GRLBNFR M A J Bone Jt Surg 41 A  
535 1959
- LIER G & SCHWENK W Werkstoffe und Korrosion 2 16 1959
- SCALFS J T J Bone Jt Surg 38 B 751 1956
- SCALES J T & ZAREK J M J Bone Jt Surg 37 B 527 1955
- SCALFS J T WINTER G D & SHIRLEY H T J Bone Jt Surg 41 B  
810 1959
- SEYFARTH H & VOIGT G Acta Histochem 1 240 1955
- SHERMAN W O Arch Surg 40 838 1940
- UBRIC H H The Corrosion Handbook John Wiley and Sons Inc  
New York USA 1948
- VENABLE C S J Bone Jt Surg 30 A 24 1948
- VENABLE C S STUCK W G & BEACH A Ann Surg 105 917 1937
- VENABLE C S & STUCK W G The International Fixation of Fractures  
Charles C. Thomas Springfield 111 1941
- ZAREK C A Metal Progress July 95 1955

AN  
LIBRARY,

COLLEGE,  
JALPUR



S M S MEDICAL COLLEGE LIBRARY  
DUE DATE SLIP

*This book is to be returned on or before the date  
marked below —*

A fine of 25 nP will be charged for each day  
the book is kept overtime

|  |  |  |  |
|--|--|--|--|
|  |  |  |  |
|--|--|--|--|







*ACTA ORTHOPAEDICA SCANDINAVICA*

SUPPLEMENTUM no 50

---

FROM THE ORTHOPAEDIC CLINIC, MALMO UNIVERSITY OF LUND  
CHIEF: DOCENT S. JENSEN

AN EXPERIMENTAL STUDY  
OF THE RATE OF FRACTURE HEALING

AS ASSESSED FROM THE TENSILE STRENGTH AND  
ST<sup>14</sup> ACTIVITY OF THE CALLUS WITH SPECIAL REFERENCE TO  
THE EFFECT OF INTRAMEDULLARY NAILING

BY

JØRGEN FALKENBERG







AN EXPERIMENTAL STUDY  
OF THE RATE OF FRACTURE HEALING

AS ASSESSED FROM THE TENSILE STRENGTH AND  
SC<sup>32</sup> ACTIVITY OF THE CALLUS WITH SPECIAL REFERENCE TO  
THE EFFECT OF INTRAMEDULLARY NAILING

BY

JØRGEN FALKENBERG





## INTRODUCTION

The physical properties of intact cortical bone are well known but less attention has been given to the characteristics of callus. Apart from a few experiments (McKEOWN et al 1932 HABLER & REISS 1936 COPP & GREENBERG 1945) by mechanical methods to determine the strength of callus during fracture healing the callus has been judged radiographically and histologically in experimental animals. As shown by HABLER & REISS (1936) and ESKELUND & PILLM (1950) however the *in vivo* methods do not give reliable data on the strength of the callus.

Several investigators (BERG & KUGELMASS 1931 KEY 1934 KEY & ODELL 1955) have tried to stimulate the formation of callus in various ways but without success. In contrast ever since his earlier publications KUNTSCHER has claimed on the basis of clinical and experimental observations that medullary nailing stimulates the formation of callus (KUNTSCHER 1958).

Although opinions founded on clinical experience differ regarding the effect of nailing most authors feel that nailing neither stimulates nor inhibits callus formation (MAATZ 1943 RIEDER & SCHUMAN 1943 LAURITZEN 1949 BOHLER 1948 SYREET 1951 FONTAINE et al 1954) only few believing the use of an intramedullary nail to stimulate callus formation (SOEIR 1946 GRANJOY & SOEIR 1955).

This lack of unanimity on the effect of intramedullary nailing on callus formation is probably due in some measure to the difficulty in collecting representative data *i.e.* sufficiently large clinical series of uniform, comparable fractures. In addition no clinical method is available for judging the degree of healing with certainty. Roentgenography will give information on the amount of callus but it yields no reliable information on the stage of healing.

In contrast to clinical experience studies in experimental animals of bones fractured as uniformly as possible suggest that nailing may stimulate the formation of callus (FITTS Jr et al 1949 GALUZZI & GIANELLI 1953 TRUETA & CAVADIAS 1955). But in these investigations the callus was judged histologically and radiographically and neither of these methods permits exact estimation of the strength of the callus.

To secure reliable information on experimental fracture healing we require a method for making comparable fractures and secondly, a method permitting assessment of the formation and quality of the callus. In clinical investigation callus is judged by its strength. This criterion for judging the quality of the callus would therefore appear rational also in experimental investigations.

The present investigation is concerned with measurement of the strength of the callus in nailed and unnailed fractures of the radius of the rabbit. Determinations were made of the absolute tensile strength (in kg) of the callus in different stages of healing. The specific tensile strength of the callus ( $\text{kg/mm}^2$ ) was calculated after measurement of the transverse area of the callus. In order to secure additional data on the biological activity of the callus most of the experimental animals received an injection of  $\text{Sr}^{85}$  after which the ash weight and the radioactivity of the fracture area and the end parts of radius were determined.

## PREVIOUS DETERMINATIONS OF THE STRENGTH OF CALLUS IN EXPERIMENTAL FRACTURES

Since the skeleton serves as an organ of support it is but natural that attempts were made to judge the physical properties of the bone by methods used in the testing of materials in industry. The first attempts to judge the strength of human bone were published by WERTHEIM in 1847. Since then numerous experiments have been carried out to measure the strength and elasticity of bone and to elucidate the mechanism of different types of fractures.

It is now known that bone is almost as strong as cast iron but only one third as heavy and much more elastic. In addition it retains its elasticity, i.e. it follows Hooke's law (the elongation of a loaded elastic body is proportional to the load) until the stress is three fourths of that necessary to break the bone and even then the deviation from the straight line is only slight (BELL 1959). According to MAJ & TOAJARI (1937) and EVANS (1958) the strength of a bone varies with the number of collagen fibres in the transverse area and the course of the fibres in relation to the axis of the bone. The greater the number of fibres and the more parallel they are to the axis of the bone the stronger the bone will be. EVANS (1958) also stated that the strength of a bone appears to depend on the number of osteons per unit of transverse area. In experiments determining the tensile strength of the bone he found that few and large osteons gave greater strength than numerous small ones for the same transverse area.

To what extent the organic and inorganic substances respectively are responsible for the physical properties of the bone is not known. However the strength of bone seems to vary with its mineral content (BELL, CHAMBERS & DAWSON 1947; WIER, BELL & CHAMBERS 1949; VOSE & KUBALA 1959) and intoxication with beta propionitrile which inhibits the formation of the ground substance but appears to have no effect on the collagen fibrils (POWSETT & SHEPARD 1954) is found to reduce the strength of bone to about one fourth of normal (BELL, OLIVE DUNBAR, GILLESPIE, IBALL & JEAN OLIVER 1957).

Little however is known concerning the mechanical properties of the callus during the course of healing of a fracture. McKEOWN, LINDSAY, HARVEY & HOWES (1932) published a study of fractures of the fibula in

To secure reliable information on experimental fracture healing we require a method for making comparable fractures and, secondly, a method permitting assessment of the formation and quality of the callus. In clinical investigations, callus is judged by its strength. This criterion for judging the quality of the callus would therefore appear rational also in experimental investigations.

The present investigation is concerned with measurement of the strength of the callus in nailed and unnailed fractures of the radius of the rabbit. Determinations were made of the absolute tensile strength (in kg) of the callus in different stages of healing. The specific tensile strength of the callus (kg/mm<sup>2</sup>) was calculated after measurement of the transverse area of the callus. In order to secure additional data on the biological activity of the callus, most of the experimental animals received an injection of Sr<sup>85</sup> after which the ash weight and the radioactivity of the fracture area and the end parts of radius were determined.

normal values within 30 day. The curve for the breaking strength in their experiments showed a steady ascent. COPP & GREENBERG's result thus strengthens the impression of the unreliability of the results reported by MCKEOWN et al.

In the experiments referred to above no attempts were made to study the elasticity of the callus. Such experiments have been made by HÄBLER & REISS (1936). They studied the breaking and torsion strength of experimental fractures of the radius, ulna and tibia of dogs. The fractures were loaded to the elastic limit with simultaneous measurement of the strain. The load noted at the elastic limit was taken as a measure of the strength of the callus. In addition the callus was weighed and the Ca content determined. The experiments were performed on fractures 50 to 90 days old and the healing was also judged radiographically and clinically. The strength of the fractures was 70—50 per cent less than that of the intact bone.

As for intact bone the curve for the deflection of callus in the experiments was linear up to the elastic limit but the ascent was more gentle particularly for fracture with poor healing.

Since the diagrams given are not true strain diagram (kg/mm deflection) it is not possible to draw any conclusions about the quality of the callus in the experimental bone compared with that of the intact control bone because of differences in the transverse area of the two bones.

HÄBLER & REISS found no correlation between the amount of Ca and the physical properties of callus and concluded that the strength of the callus is dependent not only on the mineral content but also on the bond between organic and inorganic material.

### Summary

The physical properties of intact cortical bone are well known. The strength of bone appears to vary with the number of collagen fibrils in the transverse area of the bone and with the direction of the fibrils in relation of the axis of the bone. The number of osteons in the transverse area of the bone is said to be of importance.

The strength of bone increases with its mineral content but the function of the organic substance and its effect on the strength of bone are not known.

The physical properties of callus and the changes the former undergo during repair of a fracture are not properly understood. In investigations on record the strength of callus has been estimated under almost identical experimental conditions but the results obtained are not unequivocal.

## PREVIOUS EXPERIMENTAL INVESTIGATIONS OF HEALING OF FRACTURES WITH RADIOACTIVE ISOTOPES

Most isotope studies of fracture healing have been performed on rats with fracture of the shaft of the femur tibia or the fibula and without fixation of the fracture (COPP & GREENBERG 1945 BOHR & SORENSEN 1950 KARCHER 1952, BAUER 1954 c, BAUER & CARLSSON 1955, BOHR 1955 MACDONALD LORICK & PETRIELLO 1957) MACDONALD (1958) studied mineral metabolism in unfixed fractures of the rabbit A few experiments have been performed on fractures of the rat femur fixed with a medullary nail (CARTIER DE BERNARD & LAGRANGE 1956) or on rats with a sawcut in the tibia (MARSHALL & BYRON 1945) Studies in fracture healing in man with the aid of isotopes have been performed by WENDERBERG (1961)

In experiments referred to above isotopes of calcium phosphorus or strontium were used The activity determinations were performed either by external counting over the fracture or by direct measurement of the ash

After injection the isotopes accumulate in excess amounts in the fracture region The excess uptake varies with the age of the fracture Thus in femoral fractures in rats maximal activity was observed 8-15 days after fracture when it was 2-8 times higher than in the opposite leg (BAUER 1954 c BOHR 1955) In tibial fractures in rabbits the activity maximum was reached somewhat later namely 10-20 days after the fracture (MACDONALD 1958)

The increased uptake in the fracture region subsequently diminishes but even in 50 week old fractures the activity has been found to be higher than normal (BOHR 1955)

The increased activity in the callus is accompanied by an increased activity in other parts of the fractured leg (BAUER 1954 c BOHR 1955) In animals with femoral fractures it was most pronounced in the epiphysis of the fractured femur less in the epiphysis of the tibia and least in the diaphysis of the tibia (BOHR 1955)

The rise in activity is accompanied by an increase in the ash weight of the fracture region (BAUER 1954 c BOHR 1955) The ash weight reaches its maximum on the 16-20th day thus somewhat later than the radioactivity BAUER (1954 c) found a rise of about 50% in the ash weight of

the fracture region and BOHR (1933) about 30 %. Later the ash weight decreased successively with reorganisation of the callus but in 50 week old fractures the ash weight was still increased (BOHR 1955)

In bone parts adjacent to the fracture the ash weight falls initially indicating a higher rate of bone resorption than bone formation. The resorption is earliest and strongest in the epiphysis of the fractured leg. The decrease in ash weight is considerable. According to BAUER (1954c) it is 30 % in the epiphysis of the fractured femur. The surrounding bone recovers its normal mineral content but slowly. Thus BAUER & CARLSSON (1955) found a decrease in the ash weight of the ends of the fractured femur even in 60 day old fractures.

BOHR (1955) tried to relate the degree of fracture healing to the ash weight and activity of the callus. Fracture healing was judged by the degree of mobility of the fracture. BOHR found that the increase in activity was the same whether healing was good or poor but that fractures with a high ash weight of the callus showed good healing and a good recovery of the ash weight of the epiphysis. In poorly healing fractures there was a continuous decrease in the ash weight of the epiphysis.

Observations made by CARLSSON (1952) and BAUER (1954a) have shown that only a small percentage of the calcium content of the bone is exchangeable while the bulk of the calcium is irreversibly incorporated and can be released only by resorption. It has been possible to determine these calcium fractions quantitatively (BAUER, CARLSSON & LINDQVIST 1955a).

After parenteral injection of  $\text{Ca}^{45}$  the serum activity decreases owing to the intermixture of isotopes with exchangeable Ca fractions of the skeleton to excretion and to deposition of  $\text{Ca}^{45}$  in the skeleton by accretion. The bone seeking isotopes accumulate mainly in the e parts of the skeleton where growth is most rapid (CARLSSON 1951, BAUER 1954b) and it is also in those parts of the skeleton that the isotopes are reorbed first (LEBLOND, WILKINSON, BELANGER & ROUCHON 1950, BAUER 1954d).

The activity recovered from the skeleton is an expression of the rate of accretion provided the measurement is made at a time when the effect of the exchange reaction and the resorption can be precluded. As judged by plasma activity curves in rats and rabbits (BAUER 1954c, BAUER, CARLSSON & LINDQVIST 1955b, MACDONALD 1958) the amount of activity in the exchangeable calcium fraction may be considered low a few days after injection of the isotope. At the same time also the resorption is low (BAUER, CARLSSON & LINDQVIST 1955b, MACDONALD 1958).

BAUER (1954c) showed that the increased mineral content of the callus of 8 day old fractures of the femur of the rat is incorporated by accre-

tion and not reached by resorption until after 4-5 days. It is therefore possible to correlate the radioactivity measured in the callus with the amount of bone salt formed.

### *Summary*

Experiments with bone seeking isotopes have shown that the major part of the Ca content of the skeleton is incorporated by an irreversible process (accretion) and can only be released by resorption. A few per cent of the Ca is reversibly bound and available for exchange with calcium in the body fluids.

Isotope experiments with fractures have shown that fracture healing is a generalized process and that the increased deposition of bone salts in the callus is accompanied by an increased resorption and an increased accretion of bone salt in the surrounding bone. Also the bone salt deposited in the callus is bound by an irreversible process so that the amount of radioactivity measured in the callus is related to the amount of new formed bone salt.



## MATERIAL

The material consisted of adult albino rabbit of one and the same breed. The animals were about 6 month old and weighed between 2.2 and 3.0 kg. Animals of both sex were used. The ratio between males and female was 6:1. no pregnancy was observed. The animals were fed on corn turnips and hay and in summer also on greens. During the winter months vitamin D was added to the diet. The different groups consisted of animals operated upon during different seasons of the year.

One animal died soon after the operation owing to the anaesthesia and a relatively small number of animals died under anaesthesia before the operation. These animals are not included in the material accounted for in the tables.

No deaths from infection of the wound occurred but 2 animals were killed because of infection with abscess formation. During the experimental period 21 animals died from intercurrent diseases mostly enteritis. In none of these animals were any signs of infection of the region of the wound detectable. The mortality was about 8 per cent. In addition 2 animals were rejected because of fracture of the ulna on the control side during the experimental period and 3 because the radius fractured during preparation.

A small number of animals in which the radius split during nailing were excluded. In addition 10 animals operated upon and used for preliminary experiments are not included.

The material proper consisted of 244 operated animals in which the strength of the callus was measured. In 180 of these animals determinations were made of the ash weight and of the radioactivity of the experimental bones after injection of  $\text{Sr}^{85}$ .

The experimental animals were divided into 10 groups, one of 28 animals and nine of 24 animals each.

The ages of the fractures in these groups were 10, 20, 30, 40, 50, 60, 70, 80, 100 and 120 days respectively.

The experimental animals were weighed before operation and immediately they had been killed. The control animals were weighed just before they were killed. The length of the radius was measured in all animals.

The mean values found for the various determinations are given in Table 1 and 2 a and 2 b in the appendix.

tion and not reached by resorption until after 4-5 days. It is therefore possible to correlate the radioactivity measured in the callus with the amount of bone salt formed.

### *Summary*

Experiments with bone seeking isotopes have shown that the major part of the Ca content of the skeleton is incorporated by an irreversible process (accretion) and can only be released by resorption. A few per cent of the Ca is reversibly bound and available for exchange with calcium in the body fluids.

Isotope experiments with fractures have shown that fracture healing is a generalized process and that the increased deposition of bone salts in the callus is accompanied by an increased resorption and an increased accretion of bone salt in the surrounding bone. Also the bone salt deposited in the callus is bound by an irreversible process, so that the amount of radioactivity measured in the callus is related to the amount of new formed bone salt.

surface. The radius and the ulna lie close to one another and are connected by a very narrow and firm interosseous membrane. Owing to the anatomical relationships pronation and supination movements between the bones is not possible. This implies that on osteotomy of the radius the ulna serves as a splint and prevents dislocation. The marrow cavity of the radius is hour glass shaped and narrower at a short distance above the middle of the bone.

The nail is made of stainless steel wire of 1.2–1.6 mm in diameter and 6 cm long. To be sure that the nail passes through the narrowest part of the cavity the nail is pointed.

In 15 animals i.e. in about 6 per cent of the experiments the nail showed signs of corrosion. This was always located at the tip of the nail only. The corrosion is probably ascribable to the surface of the nail having been damaged by the grinding and not always having been properly polished afterwards (BECHTOL, FERGUSON & LAING 1959).

The effect of corrosion was seen in the cortex as a dark coloured spot. The corrosion may have a toxic effect on the bone tissue as shown by JØRGENSEN (1941) and MAATZ (1948) and may cause cortical necrosis with peri- and endosteal bone formation and subsequent reorganization. Such changes might possibly affect the ash weight and thereby also the radioactivity in the C parts (see Fig. 5) of the nailed bone. Examination of the ash weight of C parts with corrosion of the nail however showed that the weight varied mainly with the length of the C part. It may therefore be assumed that the corrosion was not of such an extent as to affect the results.

#### OPERATIVE TECHNIQUE

The animals were anaesthetized with Nembutal injected intraperitoneally and placed on a special operating table in a way allowing of fixation of both forelegs so that the radius on either side could be operated upon without moving the animal.

The hair on the radial side of the foreleg was cut off with electric clippers and the skin was washed with alcohol and covered with Nobecutan and sterile gauze which was fixed to the skin by the Nobecutan.

The incision was placed in the lower half of the radial side of the foreleg. There the radius is subcutaneous and covered only by the extensor tendon. Small specially designed osteotomy hooks for protection of the ulna muscles and tendons were inserted between the two forelegs. Osteotomy of both radius was done with a fine circular saw mounted on a dental engine.

On the right side a fine burr hole was made in the lower end of the radius and a nail of suitable thickness was inserted. The left radius was used as a control leg. The wounds were sutured with thin stainless steel wire and

covered with Nobecutan. The operation caused hardly any loss of blood and the animals tolerated the operation well. In the course of a few days they were moving about unhindered in their cage.

In order to check that the nail had passed through the site of osteotomy and a sufficient distance into the upper fragment of the radius, all of the nails used were originally of the same length. The length of the radius varies only slightly and it is therefore easy to judge how far the nail has entered the marrow cavity and thereby to decide whether the nail has passed through the narrowest portion of the cavity before resistance is offered. If resistance was offered to the insertion of the nail earlier than expected, the nail was extracted and replaced by a thinner one. The projecting part of the nail — about 1 cm — was cut off flush with the bone. Only in a few animals was it necessary to replace the nail by a thinner one.

In order to decrease the effect of any infection on the results of the experiment special attention was given to sterility. Nobecutan contains a bacteriostatic agent and the incision was placed through the sterile gauze which was fastened to the skin by the Nobecutan so that all contact between the skin and the operative region was avoided.

Care was taken to place the osteotomy at about 3 cm proximal to the lower end of the radius. Especially in the early stage of this work the level of the osteotomy varied somewhat. The variation was of roughly the same order in all of the group (Table 1 appendix).

### *Preparation of radius*

The animals were killed by injection of Nembutal and air intravenously. Both forelegs were amputated, the musculature including the periosteum was removed and the nail was extracted. The length of the nail and its transverse area were noted (Table 1 appendix). It was difficult to separate the radius from the ulna, particularly in animals in which fracture healing was advanced because the callus had grown to a varying extent over on the ulna which was thus partly embedded in callus. The ulna was sawn through above and below the osteotomy and the proximal and distal parts of the ulna were removed by dividing the interosseous membrane. The remaining part of the ulna was carefully removed with fine bone forceps. In eight experimental bones the preparation caused fissures in the callus. These bones were excluded from the determinations of tensile strength. As the determination of the strength etc. was too timeconsuming to be performed on freshly removed bone, the isolated radius was stored in 5 per cent formalin solution until examined.

The formalin treatment to which all the experimental bones as well as

the bones used for determination of the normal values were exposed affects the organic components of the bones. Formalin has a tendency to decrease the tensile strength of the bone but it is not known by how much (cited EVANS 1957). On the other hand the strength in compression tests is said to decrease by 13 per cent (CALIBRIS & SMITH 1951). Since all of the bones in the present investigation were treated uniformly any effect of the formalin on the tensile strength have influenced the results in a uniform manner.

#### *Apparatus and technique for testing tensile strength*

The apparatus<sup>1)</sup> used for testing the tensile strength of the bone is illustrated in Fig. 2. It is constructed of T iron  $3.5 \text{ cm} \times 3.5 \text{ cm} \times 0.5 \text{ cm}$  and consists of a lever (A) with a fulcrum (C) at the tip of a standard (B). The lever (A) can be connected with a spring balance by means of 3 holes in the lever (A). The spring balance is connected with an extension (D) which is passing freely through the footplate (E). The spring of the balance can be loaded by turning the screw (F). The indicator of the spring balance is connected with a writing device which traces changes in load on squared paper. (I) is a weight for balancing the lever (A). The holders in which the bone is fixed are attached to the lever at (G) and to the foot plate at (H). The apparatus has a maximum capacity of 125 kg and was adjusted before use. The adjustment was done in the following way. For every hole in

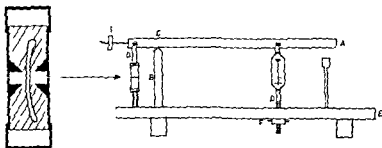


Fig. 2

Diagram of traction apparatus (see text). To left enlargement of cross section of holders with bone inserted.

<sup>1)</sup> The traction apparatus was designed by Engineer Åke I. berg, Department of Physics, University of Lund.

the lever (A) the principal balance was loaded by applying increasing loads on the lever at (G). For every load the elongation of the spring was registered by the writer connected with the spring. With these recordings a spring load elongation diagram could be plotted for each hole in the lever. From these diagrams it was possible to read the observed load necessary to break the callus since the elongation of the spring was always recorded on squared paper. During the experimental period the apparatus was repeatedly checked and no change in the elasticity of the spring was demonstrable.

In the experiments two spring balances were used. One small one permitting a maximal load of 12 kg. and a larger one permitting a maximum load of 25 kg. The smaller balance was used for the first hole in the lever for determination of the absolute tensile strength of callus in the experimental bones in the first group of animals since in that group the callus

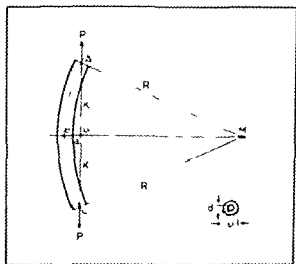


Fig. 3  
Calculation

Application of the two traction forces (I) to the slightly curved homogeneous tube cause both traction and breaking in the tube which finally breaks at (B) on increasing magnitude of the forces. If the eccentricity (BD) (a) is small in relation to the radius of the curvature of the tube (R) it is possible approximately to determine (a) from the formula  $k^2 = 2Ra$ . The force resulting in the breakdown of a tube can be calculated from the following formula

$$\text{Ultimate marginal stress} = \frac{1}{4} \frac{1}{(D^2 - d^2)} + \frac{1}{3} \frac{a}{D^2 - d^2}$$

of which the first part represents the pure traction and the second the breaking force

was not strong enough to tolerate a force necessary to produce any appreciable expansion of the larger spring.

In the present traction tests the callus was affected not only by traction but owing to the curvature of the radius also by a breaking force. However with knowledge of the transverse area of the callus as well as the radius of the curvature of the bone at the site where the force are applied it is possible to calculate the magnitude of the breaking force. This was calculated for the 60 day group of experimental bones with the use of the mean value found for the transverse area of the callus. The transverse area was assumed to be circular and the curvature of the radius in the osteotomy area was estimated to have a radius of 100 cm. The free space between the holders is 1 cm. The distance between the two points where the force (P) acts is 2 cm (Fig. 3) is assumed to be 2 cm. Using the formula (Fig. 3) below it was found that the sum of the forces acting upon the callus at the breaking moment was about 10 per cent larger than that measured in the traction apparatus.

On suspension of the holders on the traction apparatus care was taken that they were properly positioned in order to avoid an asymmetric pull. An asymmetric pull would increase the breaking force in the bone and decrease the tensile strength. The traction apparatus was balanced before application of the bone to be tested so that any traction caused by the lever and the spring balance is eliminated. The rate at which the force is applied is of importance for the tensile strength. In the traction apparatus the force acting upon the bone was produced by means of a screw arrangement which made it possible to apply the force at the same rate in all of the tests.

The holders (Fig. 2) were made of brass 2 mm thick. They consisted of two symmetrical parts each of which had the shape of a rectangular tube. The holders were provided with a metal hanger for connection to the traction apparatus. The free opening opposite the hanger is diminished by a metal wedge on either side to prevent the bone from being pulled out of the holder when loaded. The holders were designed in such a way that when they were fitted together there was a free space between them so that the part of the bone to be loaded was free.

In the beginning plaster was used as a fixative for the bone. A disadvantage of plaster however is that it is impossible to remove the bone fragments undamaged after the absolute tensile strength has been determined. In later experiments i.e. in all traction experiments in which determinations were also made of the ash weight and of the  $\text{Sr}^{85}$  activity of the bones Wood's metal which is an alloy of cadmium, zinc, lead and tin and which has a melting point of  $+67^\circ\text{C}$  was used as a fixative.

Before the bone was placed in the holders a piece of rubber was

threaded over the bone. The shape and size of the rubber sponge were adjusted to the free space between the holders and filled it when the two parts were brought together. During the fixation process the holders were fixed firmly against one another in a frame. The rubber sponge held the bone in position in the holders and at the same time prevented the molten metal from running down into the free space between the holders. As soon as the molten metal, which was poured into the holders from a small crucible, had set the holders together with the bone were placed in water and cooled and was then ready for application in the traction apparatus. As soon as the tensile strength had been determined the bone fragments were readily removed from the holders by carefully heating the latter.

In the melting process the metal was carefully heated in a crucible. If the process was regulated in such a way that there was always a little unmelted metal in the crucible the metal would never be heated above its melting point. In 20 experiments in which the bones were of the same size as the experimental bones the average temperature at the time of insertion (one temperature recorded for each right and left bone) measured thermoelectrically was  $41.9^{\circ}\text{C}$ . The temperature to which the bone was exposed on insertion was so near the physiological temperature of the rabbit which is  $39^{\circ}\text{C}$  that any effect of the temperature on the physical properties of both organic and inorganic substances of the bone may be neglected.

On insertion of the radius in the holders care was taken that the curvature of the bone was always in the same plane and that the bone was inserted vertically in the holders so that the bending force in the bone when loaded, did not vary from one experiment to another.

On loading of the bone in the traction apparatus the radius fractured at the site of osteotomy. In the most recent fracture groups (10–20–30 days) the site of the fracture still showed the smooth bone surface at the site of osteotomy surrounded by a more irregular endosteal and periosteal callus. As healing progressed the fracture surface became more irregular but the fractures retained their character of a transverse fracture throughout the experimental period.

Only in 4 cases did the radius fracture outside of the osteotomy region. These fractures occurred in three bones in the 100 day group and in one in the 120 day group. In all 4 cases it was the nailed bone that fractured in this way. The transverse areas of these bones were determined at the site of osteotomy while the values for tensile strength were excluded from the material.

#### *Measurement of cross sectional area of callus and marrow*

For determination of the area of the callus and marrow a transverse





Fig 4

Enlargement of bone callus section on photo sensitive paper

callus bone section about 2 mm thick was cut off from one of the fracture ends. Residual periosteum and marrow tissue were removed with the aid of a needle. A microscope was provided with a loupe objective and the ocular replaced by an accessory with room for a photographic cassette. By means of this apparatus a reproduction of the transverse area of the callus bone section could be obtained on photographic paper.

After development, fixation and drying of the photo-sensitive paper the part covered by the bone and the marrow respectively were cut out and the areas determined by weighing.

Here the *cross sectional area of the callus* is to be understood as the area of the total cross section of the radius at the site of oestotomy minus the marrow cavity and other cavities (see page 29) and — in nailed bones — the nail cavity.

The *cross sectional area of the marrow* is to be understood as the total cross sectional area of the marrow cavity inclusive other cavities (see page 29) and — in nailed bones — also the nail cavity.

The microscope was adjusted for tenfold enlargement (Fig 4). In the experiments it was not possible to make double determinations of the transverse area, because the callus was usually stripped on one side or the

threaded over the bone. The shape and size of the rubber sponge were adjusted to the free space between the holder and filled it when the two parts were brought together. During the fixation process the holders were fixed firmly against one another in a frame. The rubber sponge held the bone in position in the holders and at the same time prevented the molten metal from running down into the free space between the holder. As soon as the molten metal, which was poured into the holders from a small crucible, had set, the holders together with the bone were placed in water and cooled and was then ready for application in the traction apparatus. As soon as the tensile strength had been determined, the bone fragments were readily removed from the holders by carefully heating the latter.

In the melting process, the metal was carefully heated in a crucible. If the process was regulated in such a way that there was always a little unmelted metal in the crucible, the metal would never be heated above its melting point. In 20 experiments in which the bones were of the same size as the experimental bones the average temperature at the time of insertion (one temperature recorded for each right and left bone) measured thermoelectrically was  $419^{\circ}\text{C}$ . The temperature to which the bone was exposed on insertion was so near the physiological temperature of the rabbit which is  $39^{\circ}\text{C}$ . that any effect of the temperature on the physical properties of both organic and inorganic substances of the bone may be neglected.

On insertion of the radius in the holders care was taken that the curvature of the bone was always in the same plane and that the bone was inserted vertically in the holder, so that the bending force in the bone, when loaded, did not vary from one experiment to another.

On loading of the bone in the traction apparatus the radius fractured at the site of osteotomy. In the most recent fracture groups (10-20-30 days) the site of the fracture still showed the smooth bone surface at the site of osteotomy surrounded by a more irregular endosteal and periosteal callus. As healing progressed the fracture surface became more irregular but the fractures retained their character of a transverse fracture throughout the experimental period.

Only in 4 cases did the radius fracture outside of the osteotomy region. These fractures occurred in three bones in the 100-day group and in one in the 120-day group. In all 4 cases it was the naked bone that fractured in this way. The transverse areas of these bones were determined at the site of osteotomy while the values for tensile strength were excluded from the material.

#### *Measurements of cross sectional area of callus and marrow*

For determination of the area of the callus and marrow a transverse



Fig 4

Enlargement of bone callus section on photo sensitive paper

callus bone section about 2 mm thick was cut off from one of the fracture ends. Residual periosteum and marrow tissue were removed with the aid of a needle. A microscope was provided with a loupe objective and the ocular replaced by an accessory with room for a photographic cassette. By means of this apparatus a reproduction of the transverse area of the callus bone section could be obtained on photographic paper.

After development, fixation and drying of the photo sensitive paper the part covered by the bone and the marrow respectively were cut out and the areas determined by weighing.

Here the cross sectional area of the callus is to be understood as the area of the total cross section of the radius at the site of oestotomy minus the marrow cavity and other cavities (see page 29) and — in nailed bones — the nail cavity.

The cross sectional area of the marrow is to be understood as the total cross sectional area of the marrow cavity inclusive other cavities (see page 29) and — in nailed bones — also the nail cavity.

The microscope was adjusted for tenfold enlargement (Fig 4). In the experiments it was not possible to make double determinations of the transverse area because the callus was usually stripped on one side or the

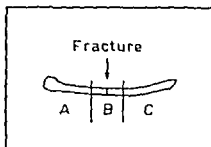


Fig 5

other during the traction experiment so that only one part of the fragments was suitable for determination of the transverse area.

The transverse area of the callus and of the marrow could be determined in this way with satisfactory accuracy. In experiments with a total of 20 determinations of the same section of the bone the mean error of the mean was found to be about 0.5 per cent. The specific tensile strength was then calculated by dividing the absolute tensile strength by the transverse area.

#### *Measurement of ash weight and radioactivity after injection of $Sr^{88}$*

In the isotope experiments each animal received a dose of 5  $\mu$ c of  $Sr^{88}$  dissolved in 1 ml of physiological saline. The dose was given intravenously 1 day before the animals were killed.

After the tensile strength and the areas had been measured the radius was divided into three parts A, B and C (Fig 5). The pieces of bone were then ashed in an electric oven at a temperature of 800–850  $^{\circ}$ C, and the ash of each of the 3 pieces was weighed on an analytical balance with an accuracy of 0.5 mg. The ash was then dissolved in concentrated nitric acid which was afterward made up to 50 ml with distilled water. The activity in the entire solution was determined. In a few instances in the beginning of the investigation only 1 ml of the solution was used for assessing the radioactivity.

The activity was measured in a scintillation counter or a scintillation well counter for the 1 ml aliquot. 10,000 counts were recorded for each specimen. The background was recorded for 3,000 counts. The counting rate of the samples varied between the groups from 2 to 20 counts per second. The background varied from 0.8 to 1.3 counts per second mainly because of variation in discriminator and HV settings of the equipment. The standard deviation of the values were less than 5 per cent of the net counts of the samples. The activity is expressed in per cent of the doses given  $\cdot 10^3$ .

### *Determination of normal values*

The 32 animal used for determinations of normal values of the bone and marrow area tensile strength, ash weight and radioactivity were of the same breed as the experimental animal and of the same weight (Table 2 a and 2 b appendix)

Normal value for bone and marrow areas were based on measurement in 16 animal of transverse sections of the radius made about 3 cm proximal to its lower end. The values are given as the means of 32 determination (16 right and 16 left) (Table 2 b appendix)

The normal values for the absolute and specific tensile strength were determined on other 16 animal. They are given as the mean of 32 determination (16 right and 16 left) (Table 2 a appendix)

The normal value for the ash weight and radioactivity were based on all of the 32 control animals and the normal value are given as mean of 64 determinations (32 right and 32 left) (Table 2 b appendix)

On estimation of the normal absolute tensile strength of the radius the intact bone was fixed in the holders in the same way as the experimental bone. That part of the radius corresponding to the site of the osteotomy in the experimental bones was placed in the free space between the holder. The purpose of the test was to fracture the intact radius at the site where the osteotomy was made in the experimental bones. But it was found that as a rule the intact radius did not fracture at the intended site but a short distance above or below the free space in the part fixed by the metal. This can be explained by the strong forces to which the intact radius was exposed were acting nearer the ends of the bone with the result that owing to the stronger curvature particularly in the proximal part of the bone the bending force arising during the test were greater than in the experimental bones.

The values found for the total and specific tensile strength of the bones must therefore be accepted with caution. The specific tensile strength of the rabbit radius in the experiments described was found to be 11.6 kg/mm<sup>2</sup> and was thus much the same than those reported in the literature where an average value of about 10 kg/mm<sup>2</sup> has been given for a variety of long bones of different animals (EVANS 1957)

### *Statistical methods*

The mean, standard deviation and standard error of the mean were calculated according to conventional formulae. The t test was used in comparing the mean. The significance is denoted by P which signifies the probability of a difference at least as large as that observed arising by chance. The difference was said to be highly significant when  $P < 0.001$ .

significant when  $0.001 < P < 0.01$  and probably significant when  $0.01 < P < 0.05$ . The  $\alpha$  level of significance are given in the text as \*\*\* \*\*, and \*, respectively.

The difference between the results in the nailed and unnailed bone was judged on the basis of the calculated ratio between nailed and unnailed bone. The significance was judged by comparison with the ratio between the normal values found for the right and left radius.

An analysis was made of the mutual relationship of the absolute tensile strength on one hand and the ash weight, activity and mineral density of the callus on the other hand.<sup>1</sup>

In order to ascertain whether any difference existed regarding the above mentioned data, each of the various groups of animals was studied separately. For this purpose the data were first studied by a qualitative method. Each experimental group was divided into two subgroups according to the strength of the callus, plus variants and minus variants, in relation to the median values. In the same way the animals were classified according to the other data into plus and minus variants. The number of experimental animals that were plus variants for both tensile strength and ash weight, for example, will indicate whether any demonstrable covariation exists.

If the number of experimental animals is  $N$ , the number of animal with plus variation regarding tensile strength is  $n_1$ , the number of animal with plus variation regarding ash weight, for example, is  $n_2$ , and the number of animals with plus variation for both properties  $n_{12}$ , and if both properties are independent of one another, the expected value of  $n_{12}$  is  $= n_1 \cdot n_2 / N$ . The limits of error can be deduced from the mean error of the difference between the observed and expected value, which is  $1/2 \sqrt{N}$ , since  $n_1$  and  $n_2$ , owing to the method of division of the groups, is practically speaking,  $1/2 \sqrt{N}$ . Comparison of the differences between the observed and calculated values for  $n_{12}$  with the mean error will show whether any demonstrable covariation exists.

The data which appeared to show a covariation, as judged by the preliminary qualitative analysis, were then studied quantitatively with calculation of the correlation coefficient, and the magnitude of the latter was compared with its mean error, which is of the order  $1/2 \sqrt{N}$ .

<sup>1</sup> The statistical analysis was performed by Prof. C. E. QUEENELL of the Institute of Statistics, University of Lund.

## RESULTS

At operation the fixation of the fragments of the bone submitted to osteotomy was distinctly better in the bone in which an intramedullary nail had been used. While a certain amount of mobility was noted at the site of osteotomy in the unnailed bones the nailed bones were rigid. On preparation the unnailed bones showed no sign of displacement of the osteotomy and pseudarthrosis was not seen in any of the experimental bone.

*Findings around the end of the nail*

The end of the nail which projected 1–2 mm beyond the cortex at the site on insertion was found to be surrounded by connective tissue on the 10th day and after about 4–6 weeks it was more or less encased in callus.

*The periosteal and endosteal callus*

The periosteal callus on the control side was found to be more or less egg shaped while on the nailed side it was spindle shaped (Fig. 6). This difference gradually disappeared and from the 50 day group on it was no longer demonstrable. On the nailed side the periosteal callus tended to extend somewhat longer proximally and distally to the osteotomy. There was a difference also in the extent of the endosteal callus between the nailed bone and the control bone. In the control bones endosteal callus formed only near the osteotomy 1–2 mm proximally and distally to the latter. In the nailed bones and then particularly radially endosteal callus extended more or less continuously along the nail.

The callus in the 10 day group was fibrous and elastic but already from the 20 day group on it was firm and hard.

*Resorption cavities in callus*

During the course of healing gross cavities of different shape, size and number were seen in the callus. The cavities were scattered so widely in the cross section of the callus that it was often not possible to distinguish endosteal from periosteal cavities with the naked eye. The cavities showed up distinctly in the photographs of the cross section of the callus on which they could be counted. The occurrence of these cavities within the different

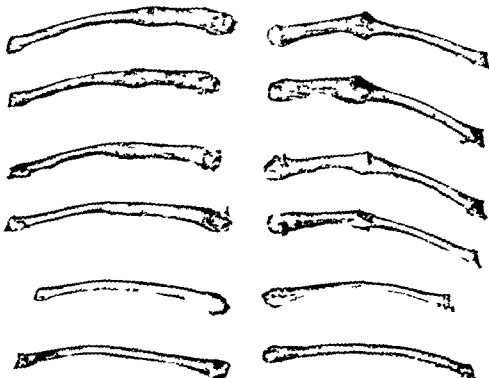


Fig. 6

Illustration of 20 day old unnailed (r) and nailed (l) fractures and of normal bones (l-r)

| age<br>of<br>fracture<br>in day | No. of<br>animal | nailed                            | unnailed                            |
|---------------------------------|------------------|-----------------------------------|-------------------------------------|
|                                 |                  | No. of<br>bones<br>with<br>cavity | No. of<br>bones<br>with<br>cavities |
| 10                              | 24               | 0                                 | 7 (0)                               |
| 20                              | 28               | 0                                 | 0                                   |
| 30                              | 24               | 1 (0)                             | 4 (0)                               |
| 40                              | 24               | 3 (0)                             | 14 (0)                              |
| 50                              | 24               | 2 (0)                             | 23 (3)                              |
| 60                              | 24               | (3)                               | 24 (0)                              |
| 70                              | 24               | (3)                               | 24 (10)                             |
| 80                              | 24               | 10 (3)                            | 24 (4)                              |
| 100                             | 24               | 12 (5)                            | 24 (3)                              |
| 120                             | 24               | 1 (6)                             | 24 (4)                              |

Figure in brackets indicate number of  
bones with more than one cavity

Table I



groups of the material is given in Table 1 which shows that cavity formation was more pronounced in the control bones

From the 60 day group cavities were found in all control bones. In the beginning the cavities were solitary but then appeared in varying number. Later the cavities tended to become solitary again i.e. the marrow cavity was reformed (See photo of cross section of bone in appendix). In the nailed bones where there was a nail cavity which cannot be regarded as a cavity proper the cavities tended to develop later than on the control side and the number of cavities was smaller (See Table 1). In the 60 day group it was however possible to observe that the nail cavity began to grow in size and to assume a more irregular outline and in some of the bone the nail cavity was surrounded by one or more small cavities. At the end of the experimental period and in contradistinction to the control side the nailed bone showed not a solitary marrow cavity but usually a nail cavity or rests of such a cavity surrounded by one or more other cavities (See photo in appendix)

#### *Defects in radius and ulna (See Fig 7)*

In the later experimental groups (70 80 100 and 120 day group) it was found in some of the bone that the marrow cavities of the radius and of the ulna had fused at the site of osteotomy. Thus a bone defect occurred in the cortex of both bones. While it was otherwise possible on preparation to distinguish the cortex of the ulna from the callus of the radius in the defects the cortex of the two bones merged. The defects were irregular and about  $1\frac{1}{2}$  mm in size. They consisted either of a smooth edged hole or more frequently a cortex defect filled with loose spongy bone. The defects were seen in both nailed and control bones with largely equal frequency. They were first observed in some bones in the 70 day group but afterwards occurred in about one half of three fourths of the bones in the 100 and 120 day groups (See Table 2)

The defects complicates the determination of the cross sectional area of the callus and the marrow because when attempts were made to saw off a slice of bone including the defect in the bone the bone split. The slice of bone used for measurement of the cross sectional area therefore included a small part of the contiguous intact bone so that the defects do not show up on photographs of the cross sectional area of bone. The cross sectional area of the marrow was therefore in reality somewhat larger than that indicated by the measurement while the area of the bone was smaller. Therefore the specific tensile strength measured will be too small in relation to that of normal bone. The error thereby introduced will however be small (7-8 %) compared with the total decrease in absolute ten-

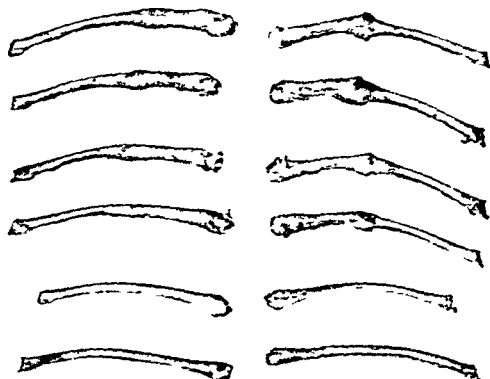


FIG. 6

Illustrations of 2) day old unnailed (r) and nailed (l) fractures and of normal bones (below)

| Age<br>of<br>fracture<br>in days | No. of<br>animals | nailed                              |     | unnailed                            |      |
|----------------------------------|-------------------|-------------------------------------|-----|-------------------------------------|------|
|                                  |                   | No. of<br>bones<br>with<br>cavities |     | No. of<br>bones<br>with<br>cavities |      |
| 10                               | 24                | 0                                   |     | 0                                   | (0)  |
| 20                               | 23                | 0                                   |     | 0                                   |      |
| 30                               | 24                | 1                                   | (0) | 4                                   | (0)  |
| 40                               | 24                | 3                                   | (0) | 17                                  | (0)  |
| 50                               | 24                | 2                                   | (0) | 23                                  | (3)  |
| 60                               | 2                 | (2)                                 |     | 24                                  | (0)  |
| 70                               | 4                 | (2)                                 |     | 4                                   | (10) |
| 80                               | 4                 | 10                                  | (3) | 4                                   | (4)  |
| 100                              | 24                | 12                                  | ( ) | 6                                   | (3)  |
| 120                              | 4                 | 12                                  | (6) | 4                                   | (4)  |

Figures in brackets indicate number of bones with more than one cavity

groups of the material is given in Table 1 which shows that cavity formation was more pronounced in the control bones

From the 60 day group cavities were found in all control bones. In the beginning the cavities were solitary but then appeared in varying number. Later the cavities tended to become solitary again i.e. the marrow cavity was re formed (See photo of cross section of bone in appendix). In the nailed bones where there was a nail cavity which cannot be regarded as a cavity proper the cavities tended to develop later than on the control side and the number of cavities was smaller (See Table 1). In the 60 day group it was however possible to observe that the nail cavities began to grow in size and to assume a more irregular outline and in some of the bones the nail cavity was surrounded by one or more small cavities. At the end of the experimental period and in contradistinction to the control side the nailed bone showed not a solitary marrow cavity but usually a nail cavity or rests of such a cavity surrounded by one or more other cavities (See photo in appendix).

#### *Defects in radius and ulna (See Fig. 7)*

In the later experimental group (70 80 100 and 120 day group) it was found in some of the bones that the marrow cavities of the radius and of the ulna had fused at the site of osteotomy. Thus a bone defect occurred in the cortex of both bones. While it was otherwise possible on preparation to distinguish the cortex of the ulna from the callus of the radius in the defects the cortex of the two bones merged. The defects were irregular and about  $1 \times 2$  mm in size. They consisted either of a smooth edged hole or more frequently a cortex defect filled with loose spongio. The defects were seen in both nailed and control bones with largely equal frequency. They were first observed in some bones in the 70 day group but afterwards occurred in about one half of three fourths of the bones in the 100 and 120 day groups (See Table 2).

The defects complicate the determination of the cross sectional areas of the callus and the marrow because when attempts were made to saw off a slice of bone including the defect in the bone the bone split. The piece of bone used for measurement of the cross sectional area therefore included a small part of the contiguous intact bone so that the defects do not show up on photographs of the cross sectional area of bone. The cross sectional area of the marrow was therefore in reality somewhat larger than that indicated by the measurements while the area of the bone was smaller. Therefore the specific tensile strength measured will be too small in relation to that of normal bone. The error thereby introduced will however be small (7-8 %) compared with the total decrease in absolute

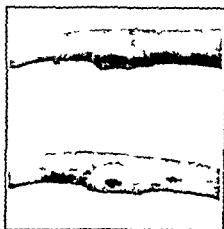
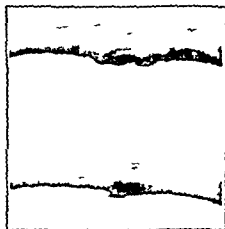
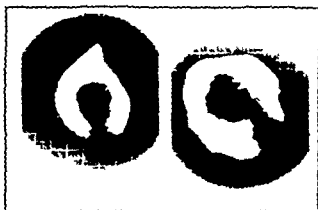


Fig. 7

Bones with defect. Above - cross section

| age<br>of<br>fracture<br>in days | No. of<br>animals | nailed   | unnailed                                       |
|----------------------------------|-------------------|--|--|
|                                  |                   | No. of<br>bones<br>with<br>cortical<br>defects | No. of<br>bones<br>with<br>cortical<br>defects |
| 0                                | 24                | 2  | 2  |
| 80                               | 24                | 1  | 4  |
| 100                              | 24                | 12   | 18   |
| 120                              | 24                | 16   | 18   |

Table 2

the strength owing to the defect. The influence of the defect on the tensile strength can be approximately calculated (See Fig. 8). If the size of the defect is taken as 1 mm  $\times$  2 mm on the average and if the mean value of the transverse area of the marrow and the bone in the 100-day group

of experimental animals be used the tensile strength calculated according to the formulae below will be about 40 % less than it would have been if the radius had been without defect

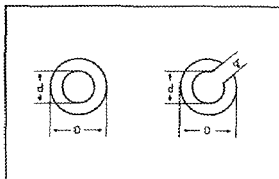


Fig 8

Fig 8 In a straight homogeneous tube exposed to pull of P the following tension will arise

$$\sigma = \frac{P}{\frac{\pi}{4} (D^2 - d^2)}$$

In a corresponding tube with a hole with a diameter (b) is subjected to the same pull (P) the tension at the edge of the hole will be

$$\sigma = \frac{P}{\frac{\pi}{4} (D^2 - d^2) - b \left( \frac{D - d}{2} \right)} + \frac{P \cdot e}{W}$$

the first member representing the tension and the second the bending force. In the above formula (e) is the eccentricity and can be calculated from the formula

$$e = \frac{\frac{\pi}{4} (D^2 - d^2) \frac{D - d}{2} - b \left( \frac{D - d}{2} \right) \left( \frac{D - d}{4} \right)}{\frac{\pi}{4} (D^2 - d^2) - b \left( \frac{D - d}{2} \right)} \cdot \frac{D}{2}$$

and the resistance (W) can be calculated from the formula

$$W = \frac{D}{2} \left[ \frac{\pi}{64} (D^4 - d^4) + \frac{\pi}{4} (D^2 - d^2) e^2 - \frac{1}{12} b \left( \frac{D - d}{2} \right)^2 - b \frac{D - d}{2} \left( \frac{D - d}{4} + \frac{d}{2} + e \right)^2 \right]$$

The tension will be (1) for the cross section without any defect

$$\sigma = \frac{1}{F_1} P$$

for a transverse area of (F) and (2) for a cross section with a defect

$$\sigma = \frac{P}{F_1} + \frac{M}{W}$$

where (F<sub>1</sub>) is the transverse area minus the area of the defect and M = P · e

### Estimation of tensile strength

The absolute tensile strength (Fig 9 and Table) of both experimental bones increased from the 10th day. The increase was almost linear during the first two thirds of the experimental period. From about the 40th day the absolute strength on the control side showed a tendency to be above that on the nailed side and about the 60th day the absolute strength of the control bones was higher than that of the nailed bones(\*\*\*)

In the further course no difference was found in the absolute tensile strength, though the strength of the control bone continued to show a tendency to be higher than on the nailed side. After the 80th day the increase in strength was less rapid.

The absolute strength on the 10th day was about 4 % of normal, on the 30th day it rose to 20 % and on the 80th day it was about 30 % of normal. The absolute strength on the 60th day was about 30 % higher on the control side than on the nailed side.

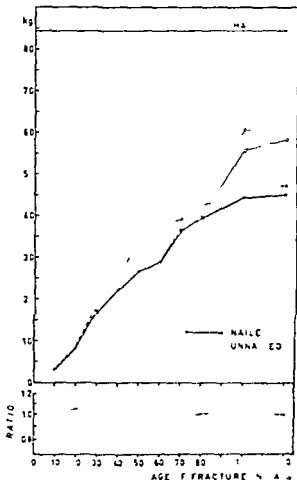


Fig 9

Variation in absolute tensile strength with age of fracture. Thin lines indicate values corrected for defects in bones.

Below: Curve for ratio between nailed and unnailed bones, in log scale.

The data forming the basis of the curves are given in tabular form below.

| age of fracture in days     | absolute tensile strength in kg |      |      |               |               |      |      |               | ratio absolute tensile strength nailed/unnailed |      |      |               |
|-----------------------------|---------------------------------|------|------|---------------|---------------|------|------|---------------|---|------|------|---------------|
|                             | nailed                          |      |      |               | unnailed      |      |      |               |   |      |      |               |
|                             | No of animals                   | mean | s d  | error of mean | No of animals | mean | s d  | error of mean | No of animals                                   | mean | s d  | error of mean |
| 10                          | 22                              | 3.1  | 1.0  | 0.21          | 24            | 3.2  | 1.0  | 0.20          | 22  | 1.01 | 0.40 | 0.083         |
| 20                          |                                 | 8.1  | 3.3  | 0.63          | 27            | 8.1  | 3.4  | 0.63          | 26  | 1.06 | 0.43 | 0.084         |
| 30                          | 24                              | 17.1 | 5.6  | 1.14          | 24            | 18.5 | 7.4  | 1.53          | 24  | 1.08 | 0.64 | 0.130         |
| 40                          | 24                              | 27.1 | 8.0  | 1.63          | 4             | 23.4 | 8    | 1.77          | 4   | 0.95 | 0.4  | 0.085         |
| 50                          | 24                              | 26.7 | 7.4  | 1.57          | 23            | 34.4 | 11.2 | 2.8           | 24  | 0.86 | 0.39 | 0.080         |
| 60                          | 24                              | 29.0 | 11   | 2.8           | 24            | 38.2 | 12.8 | 2.61          | 4   | 0.74 | 0.23 | 0.044         |
| 70                          | 23                              | 36.5 | 12.0 | 2.49          | 24            | 39.3 | 8.1  | 1.64          | 23  | 0.94 | 0.9  | 0.061         |
| 80                          | 22                              | 39.6 | 10.9 | 2.31          | 24            | 47.4 | 10.1 | 2.07          | 22  | 1.00 | 0.4  | 0.090         |
| 100                         | 21                              | 45.8 | 13.3 | 2.89          | 24            | 4.8  | 14.9 | 3.05          | 21  | 1.03 | 0.47 | 0.092         |
| 120                         | 23                              | 46.4 | 14.9 | 2.95          | 23            | 4.3  | 12.6 | 63            | 2   | 0.99 | 0.29 | 0.061         |
| mean normal values 31 bones |                                 | 84.2 | 13.3 | 2.35          |               |      |      |               |   | 1.02 | 0.15 | 0.037         |

The specific tensile strength (See Fig. 10 and Table) of both experimental bones increased from the 10th day. Like the absolute strength the specific tensile strength increased almost linearly during the first two thirds of the experimental period. About the 20th day the specific strength was greater on the nailed side (\*\*\*) From then on no difference was found in the strength on the two sides until about the 60th day when the specific strength on the control side was greater(\*\*). The increase in strength was then less rapid and no subsequent difference was found between the specific strength on the two sides.

The specific strength on the 10th day was about 1 % of normal. On the 30th day it was about 6 % and on the 80th day about 20 % of normal.

On the 20th day the specific strength on the nailed side was more than 50 % greater than on the control side while on the 60th day it was about 40 % greater on the control side than on the nailed side.

#### *Estimation of cross sectional area of callus and marrow*

The cross sectional area of the callus (Fig. 11 and Table) increased from the beginning of the experimental period in both the nailed bones and the control bones. The increase on either side reached a maximum on about the 20th day after which the cross sectional area decreased on both sides. On the 10th day there was no significant difference between the area on either side but on the 20th and 30th days the area on the

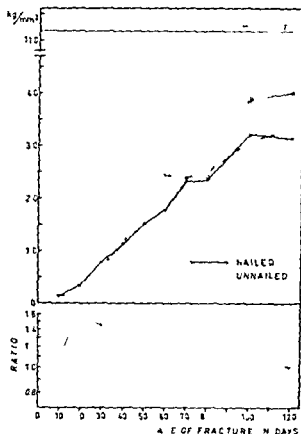


Fig 10

Variation in the specific tensile strength with age of fracture

Thin lines indicate values corrected for defects in bone

Below Curve for ratio between nailed and unnailed bones given in log scale

N.B. Scale broken between 10 and 40 kg/mm<sup>2</sup>

The data forming the basis of the curves are given in tabular form below

| age of fracture in days    | specific tensile strength in kg. mm <sup>2</sup> |       |       |               |                |      |       |               | ratio of specific tensile strength nailed/unnailed |      |       |               |       |
|----------------------------|--|-------|-------|---------------|----------------|------|-------|---------------|--|------|-------|---------------|-------|
|                            | nailed   |       |       |               | unnailed       |      |       |               |  |      |       |               |       |
|                            | No. of animals                                   | mean  | s. d. | error of mean | No. of animals | mean | s. d. | error of mean | No. of animals                                     | mean | s. d. | error of mean |       |
| 10                         | 2  | 0.14  | 0.06  | 0.012         | 24             | 0.13 | 0.04  | 0.010         | 2  | 1.11 | 0.38  | 0.081         |       |
| 20                         | -  | 0.34  | 0.15  | 0.039         | 24             | 0.25 | 0.09  | 0.017         | 6  | 1.6  | 0.4   | 0.040         |       |
| 30                         | 4  | 0.8   | 0.33  | 0.069         | 4              | 0.68 | 0.33  | 0.063         | 24   | 1.46 | 1.09  | 0.27          |       |
| 40                         | 24   | 1.13  | 0.49  | 0.099         | 24             | 1.14 | 0.50  | 0.10          | 24   | 1.09 | 0.48  | 0.093         |       |
| 50                         | 24   | 1.5   | 0.4   | 0.086         | 24             | 1.2  | 0.4   | 0.11          | 24   | 1.0  | 0.39  | 0.10          |       |
| 60                         | 24   | 1.78  | 0.65  | 0.132         | 24             | 2.44 | 0.86  | 0.16          | 24   | 0    | 0.27  | 0.055         |       |
| 70                         | 23   | 2.33  | 0.60  | 0.121         | 24             | 2.39 | 0.65  | 0.13          | 23   | 1.03 | 0.35  | 0.07          |       |
| 80                         | --   | 3.36  | 0.51  | 0.109         | 24             | 2.51 | 0.59  | 0.11          | --   | 1.00 | 0.33  | 0.063         |       |
| 100                        | 21   | 3.22  | 1.05  | 0.20          | 24             | 3.10 | 1.36  | 0.20          | 21   | 1.12 | 0.53  | 0.114         |       |
| 120                        | 23   | 3.13  | 0.91  | 0.189         | 23             | 3.29 | 0.7   | 0.203         | 22   | 1.00 | 0.38  | 0.081         |       |
| mean normal value 31 bones |  | 11.16 | 12.1  | 0.2           |                |      |       |               |  |      | 0.99  | 0.18          | 0.016 |



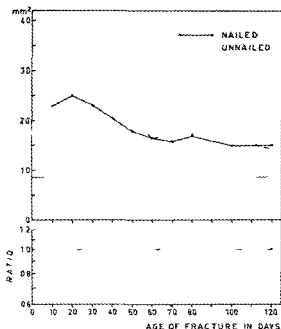


Fig 11

Variation in cross sectional area of callus with age of fracture

Below Curve for ratio between nailed and unnailed bones given in log scale

The data forming the basis of the curves are given in tabular form below

| age of fracture in days     | cross section of callus in mm <sup>2</sup> |      |     |               |               |      |     |               | ratio cross section of callus nailed/unnailed |      |      |               |
|-----------------------------|--|------|-----|---------------|---------------|------|-----|---------------|---|------|------|---------------|
|                             | nailed                                     |      |     |               | unnailed      |      |     |               |   |      |      |               |
|                             | No of animals                              | mean | s d | error of mean | No of animals | mean | s d | error of mean | No of animals                                 | mean | s d  | error of mean |
| 10                          | 24   | 28   | 6.3 | 1.28          | 24            | 25.0 | 5.6 | 1.15          | 24  | 0.93 | 0.22 | 0.045         |
| 20                          | 28   | 24.9 | 6.8 | 1.19          | 28            | 36.7 | 8.7 | 1.64          | 28  | 0.70 | 0.22 | 0.041         |
| 30                          | 24   | 30   | 4.6 | 0.93          | 24            | 28.7 | 5.2 | 1.06          | 24  | 0.82 | 0.18 | 0.037         |
| 40                          | 24   | 35   | 5.3 | 1.08          | 24            | 23.6 | 5.0 | 1.02          | 24  | 0.89 | 0.24 | 0.049         |
| 50                          | 24   | 17.9 | 4.8 | 0.58          | 24            | 20.5 | 4.4 | 0.89          | 24  | 0.90 | 0.00 | 0.041         |
| 60                          | 24   | 16.4 | 3.5 | 0.72          | 24            | 16.4 | 5.1 | 1.03          | 24  | 1.04 | 0.22 | 0.044         |
| 70                          | 4  | 15.8 | 3.1 | 0.62          | 24            | 17.2 | 4.2 | 0.85          | 24  | 0.95 | 0.22 | 0.045         |
| 80                          | 24   | 16.9 | 3.4 | 0.69          | 24            | 17.1 | 3.1 | 0.64          | 24  | 1.00 | 0.21 | 0.043         |
| 100                         | 1  | 14.9 | 3.8 | 0.81          | 24            | 16.3 | 4.0 | 0.81          | 23  | 0.95 | 0.14 | 0.030         |
| 120                         | 23   | 15.0 | 3.2 | 0.64          | 24            | 15.2 | 3.4 | 0.69          | 23  | 1.01 | 0.16 | 0.033         |
| mean normal values 12 bones |  | 8.6  | 1.2 | 0.1           |               |      |     |               |   | 0.99 | 0.08 | 0.000         |

control side was about 50 % and 25 % respectively, larger than on the nailed side(\*\*\*). From about the 40th day and during the rest of the experimental period there was no significant difference in the area on the two sides.

The transverse area of the radius at the site of osteotomy increased considerably because of the callus formation. When the callus reached its maximum the cross sectional area of the nailed bone was almost 3 times normal and that on the control side about 1 1/2 times normal. The reduction in the area as a consequence of reorganization of the callus was remarkable. Thus between the 20th and 60th days the area diminished on the nailed side by about 35 % and on the control side by about 55 %. During the last half of the experimental period the cross sectional area decreased only slightly and to the same extent on both sides. At the end of the experimental period the cross sectional area on both sides was about twice as large as that of a normal radius.

*The cross sectional area of marrow* (Fig. 12 and Table) on the control side was markedly reduced already on the 10th day because of the formation of endosteal callus and on the 20th day it was 0 i.e. the marrow in all of the unnailed bones was filled with endosteal callus. On the 30th day the marrow showed signs of restoration and on the 60th day the transverse area of the marrow was of normal size. The area of the marrow increased until the 70th day and was then larger than normal(\*\*\*), and retained this size during the rest of the experimental period. On the 120th day the marrow cavity consisted of a solitary cavity in nearly all of the bones in the control group (see photograph in appendix) and the area of the marrow was then increased by 50 % in relation to that of normal marrow.

Also on the nailed side the marrow cavity was reduced in size by the formation of endosteal callus. (See photograph in appendix). Since the nail filled part of the marrow cavity the amount of endosteal callus was not so large as on the control side. While the area of the marrow on the control side from the 30th day grew rapidly, the area of the marrow on the nailed side remained unchanged until the 60th day, reached normal values on the 70th day, and showed a tendency to lie above normal after the 80th day.

#### *Measurements of ash weight and radio activity*

*The ash weight of the B parts* (See Fig. 13 and Table) on the 10th day was increased on the nailed side and on the control side(\*). The ash weight then rose on both sides to reach a maximum on the 30th day. It then decreased on both sides, first rapidly and then slowly during the rest of the experimental period. On the 10th day there was no difference in ash

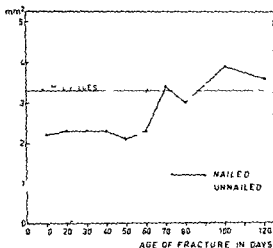


Fig. 1<sup>a</sup>

Variation in cross sectional area of marrow with age of fracture. The data forming the basis of the curve are given in tabular form below.

| age of fracture in days     | cross section of marrow in mm <sup>2</sup> |      |      |               |               |      |      |               |
|-----------------------------|--|------|------|---------------|---------------|------|------|---------------|
|                             | nailed                                     |      |      |               | unnailed      |      |      |               |
|                             | No of animals                              | mean | s.d. | error of mean | No of animals | mean | s.d. | error of mean |
| 10                          | 21   | 2.2  | 1.0  | 0.20          | 24            | 0.4  | 0.9  | 0.18          |
| 20                          | 8  | 3    | 1.1  | 0.31          | 28            | 0    | 0    | 0             |
| 30                          | 24   | 2.3  | 0.9  | 0.17          | 24            | 0.2  | 0.6  | 0.13          |
| 40                          | 24   | 2.3  | 0.8  | 0.17          | 4             | 1.3  | 1.1  | 0.3           |
| 50                          | 4  | 2.1  | 0.8  | 0.16          | 24            | 2.2  | 1.1  | 0             |
| 60                          | 24   | 2.3  | 1.2  | 0.24          | 24            | 3.2  | 1.5  | 0.30          |
| 70                          | 24   | 3.4  | 1.9  | 0.38          | 24            | 4.6  | 1.6  | 0.33          |
| 80                          | 24   | 3.0  | 1.5  | 0.30          | 24            | 4.0  | 1.0  | 0.19          |
| 100                         | 21   | 3.9  | 1.3  | 0.28          | 24            | 4.4  | 1.3  | 0.26          |
| 120                         | 23   | 3.6  | 1.7  | 0.35          | 24            | 4.9  | 1.4  | 0.3           |
| mean normal values 32 bones |  | 3.3  | 0.9  | 0.16          |               |      |      |               |

weight between the two sides. On the 20th through 40th days the ash weight on the control side was found to be larger than on the nailed side<sup>\*\*</sup>. The increase in relation to the nailed side varied from about 15 % to 10 %. Afterwards no difference was found in ash weight between the two sides until the 120th day when the ash weight of the nailed side was greater<sup>(\*\*)</sup>.

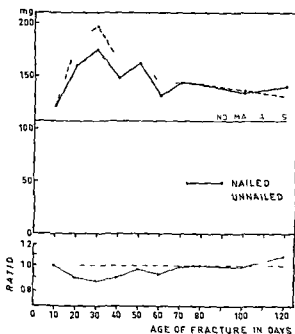
The increase in ash weight of the B parts in relation to normal values was considerable. When the ash weight of the experimental B parts was maximal the increase on the nailed side was about 65 % while that on the

Fig 13

Variation in the ash weight of the B parts with age of fracture

Below Curve for ratio between nailed and unnailed bones given in log scale

The data forming the basis of the curves are given in tabular form below



| age of fracture in days       | No of animals | wt of bone a h in mg |     |               |          |     |               | ratio Wt of bone a h nailed/unnailed |      |               |
|-------------------------------|---------------|----------------------|-----|---------------|----------|-----|---------------|--------------------------------------|------|---------------|
|                               |               | nailed               |     |               | unnailed |     |               |                                      |      |               |
|                               |               | mean                 | s d | error of mean | mean     | s d | error of mean | mean                                 | s d  | error of mean |
| 10                            | 17            | 122                  | 34  | 8.2           | 121      | 26  | 6.4           | 1.00                                 | 0.13 | 0.032         |
| 20                            | 16            | 160                  | 20  | 5.0           | 184      | 37  | 9.2           | 0.90                                 | 0.00 | 0.001         |
| 30                            | 15            | 175                  | 30  | 7.9           | 197      | 35  | 8.9           | 0.87                                 | 0.15 | 0.039         |
| 40                            | 16            | 148                  | 32  | 7.9           | 161      | 31  | 7.7           | 0.90                                 | 0.13 | 0.031         |
| 50                            | 16            | 162                  | 36  | 8.9           | 167      | 32  | 8.0           | 0.97                                 | 0.12 | 0.031         |
| 60                            | 18            | 131                  | 31  | 7.4           | 141      | 36  | 8.5           | 0.93                                 | 0.13 | 0.029         |
| 70                            | 17            | 144                  | 30  | 7.3           | 144      | 24  | 5.8           | 0.99                                 | 0.10 | 0.004         |
| 80                            | 17            | 142                  | 22  | 5.0           | 143      | 17  | 4.2           | 1.00                                 | 0.17 | 0.042         |
| 100                           | 24            | 134                  | 25  | 5.0           | 136      | 21  | 4.2           | 0.99                                 | 0.13 | 0.007         |
| 120                           | 24            | 140                  | 29  | 6.0           | 130      | 21  | 4.3           | 1.09                                 | 0.18 | 0.037         |
| mean normal values 64 B parts |               | 107                  | 14  | 1.8           |          |     |               | 0.98                                 | 0.07 | 0.012         |

control side was about 85 %. The ash weight of the B parts remained increased during the entire experimental period. The increase in the ash weight at the end of the experimental period was about 25 %.

*The radioactivity of the B parts* (See Fig 14 and Table) of both experimental bones were higher than normal already on the 10th day(\*\*\*) The activity then rose on both sides to reach a peak on about the 20th day. Afterwards the activity decreased first rapidly and then slowly and even at the end of the experimental period it was larger than normal on both sides.

On the 10th day the activity was higher on the nailed side( ) The difference was about 12 %. On the 20th day the activity was 35 % higher on the control side than on the nailed side(\*\*\*) Between the 30th and 70th days no difference in activity was found between the two sides. After the 70th day the activity on the nailed side tended to be higher than on the control side and on the 80th day and 120th day it was higher( ) The activity on the 80th day and 120th day was about 15 % and 8 % respectively higher than on the control side.

The increase in activity compared with normal values was considerable. When the activity was maximal it was 5.8 times normal on the nailed side and 8.3 times normal on the control side. At the end of the experimental period the activity on both sides was still some 1.7 times normal.

*The ash weight of the A parts* (See Fig 15 and Table) on the control side was lower than normal on the 10th day(\*\*) and then increased to reach normal on the 20th day. The ash weight stayed about normal until it rose about the 70th day and remained higher than normal(\*\*) through the end of the experimental period. The initial loss in ash weight measured on the 10th day was about 15 %. On the 80th day the ash weight was about 10 % higher than normal.

The ash weight of the nailed bones was normal on the 10th day. It then rose and on the 30th day it was higher than normal(\*\*). The ash weight fluctuated between the 30th day and the 70th days about the 50th day it was increased(\*) while about the 40th and 60th days it lay within the normal range. From the 70th day and throughout the rest of the experimental period it remained higher than normal(\*\*). The increase in weight on the 30th day was about 12 % and on the 80th day about 20 % and on the 120th day about 13 % above normal.

The ash weight of the A parts of the nailed bones was higher than on the control side throughout the experimental period except on the 40th day. The increase in ash weight in relation to the control side was about 15 % on the 10th day and then varied during the rest of the experimental period and on the 120th day it was about 6 %.

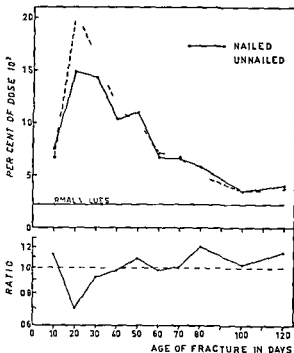


Fig 14

Variation in the activity of the B parts with age of fracture

Below Curve for ratio between nailed and unnailed bones given in log scale

The data forming the basis of the curves are given in tabular form below

| age of fracture in day        | No of animals | Sr <sup>88</sup> activity in % of dose $\times 10^3$ |     |               |          |     |               | ratio Sr <sup>88</sup> activity nailed/unnailed |      |               |
|-------------------------------|---------------|--|-----|---------------|----------|-----|---------------|---|------|---------------|
|                               |               | nailed   |     |               | unnailed |     |               |   |      |               |
|                               |               | mean   | s d | error of mean | mean     | s d | error of mean | mean  | s d  | error of mean |
| 10                            | 17            | 7.5  | 2.6 | 0.63          | 6.7      | 1.9 | 0.47          | 1.13  | 0.27 | 0.066         |
| 20                            | 16            | 14.9   | 8.6 | 2.16          | 20.4     | 9.9 | 2.47          | 0.70  | 0.14 | 0.036         |
| 30                            | 15            | 14.3   | 6.8 | 1.75          | 16.0     | 6.8 | 1.76          | 0.92  | 0.27 | 0.070         |
| 40                            | 16            | 10.3   | 3.4 | 0.86          | 11.2     | 3.3 | 0.82          | 0.98  | 0.21 | 0.053         |
| 50                            | 16            | 11.0   | 6.3 | 1.57          | 10.1     | 4.5 | 1.11          | 1.09  | 0.29 | 0.072         |
| 60                            | 18            | 6.7  | 2.4 | 0.57          | 7.1      | 3.1 | 0.73          | 0.98  | 0.24 | 0.057         |
| 70                            | 17            | 6.6  | 2.5 | 0.60          | 6.7      | 2.8 | 0.67          | 1.01  | 0.24 | 0.058         |
| 80                            | 17            | 5.8  | 2.6 | 0.63          | 5.0      | 2.1 | 0.50          | 1.21  | 0.48 | 0.117         |
| 100                           | 24            | 3.5  | 1.5 | 0.31          | 3.5      | 1.4 | 0.29          | 1.03  | 0.22 | 0.045         |
| 120                           | 24            | 4.0  | 1.6 | 0.33          | 3.7      | 1.8 | 0.38          | 1.15  | 0.20 | 0.041         |
| mean normal values 64 B parts |               | 2.2  | 0.8 | 0.10          |          |     |               | 1.00  | 0.10 | 0.017         |

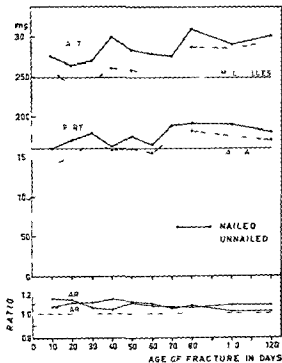


Fig 1b

Variation in a h weight of the A and C-part with age of fracture

Below Curve for ratio between  
nailed and unnailed bones given  
in log scale

The data forming the basis of curve are given in tabular form below

| age<br>of<br>fracture<br>in<br>days | No of<br>animals | wt of bone & h m g |     |                     |          |     |                     | ratio wt of bone & h |      |                     |
|-------------------------------------|------------------|--------------------|-----|---------------------|----------|-----|---------------------|----------------------|------|---------------------|
|                                     |                  | nailed             |     |                     | unnailed |     |                     | nailed unnailed      |      |                     |
|                                     |                  | mean               | s d | error<br>of<br>mean | mean     | s d | error<br>of<br>mean | mean                 | s d. | error<br>of<br>mean |
| 10                                  | 1                | 160                | .6  | 6.3                 | 138      | 19  | 4.7                 | 1.16                 | 0.09 | 0.022               |
| 20                                  | 16               | 1.1                | 2   | 5.6                 | 151      | 14  | 3.6                 | 1.14                 | 0.14 | 0.031               |
| 30                                  | 15               | 1.9                | 26  | 6                   | 168      | 28  | 7.3                 | 1.07                 | 0.14 | 0.03                |
| 40                                  | 16               | 163                | 24  | 5.9                 | 158      | 20  | 5.0                 | 1.04                 | 0.17 | 0.042               |
| 50                                  | 16               | 176                | 3   | 9.2                 | 160      | 36  | 8.9                 | 1.11                 | 0.15 | 0.038               |
| 60                                  | 18               | 164                | 27  | 6.3                 | 154      | 37  | 7.6                 | 1.08                 | 0.14 | 0.034               |
| 70                                  | 1                | 188                | 30  | 7.3                 | 155      | 23  | 5.5                 | 1.07                 | 0.10 | 0.024               |
| 80                                  | 17               | 192                | 43  | 10.3                | 183      | 3   | 8.9                 | 1.06                 | 0.16 | 0.038               |
| 100                                 | 24               | 190                | 42  | 8.7                 | 177      | 38  | 7                   | 1.09                 | 0.19 | 0.039               |
| 120                                 | 24               | 181                | 42  | 8.5                 | 170      | 42  | 8.6                 | 1.08                 | 0.16 | 0.033               |
| none normal values<br>64 & parts    |                  | 160                | 71  | 2.8                 |          |     |                     | 0.99                 | 0.03 | 0.006               |

|                    |    |     |    |      |     |    |      |      |      |       |   |
|--------------------|----|-----|----|------|-----|----|------|------|------|-------|---|
| 10                 | 17 | 275 | 46 | 11.2 | 258 | 49 | 11.8 | 1.07 | 0.06 | 0.015 | A |
| 20                 | 16 | 264 | 28 | 6.9  | 240 | 30 | 7.6  | 1.11 | 0.11 | 0.024 |   |
| 30                 | 15 | 269 | 52 | 13.3 | 244 | 49 | 12.7 | 1.11 | 0.11 | 0.027 |   |
| 40                 | 16 | 300 | 52 | 13.0 | 262 | 45 | 11.2 | 1.15 | 0.14 | 0.035 |   |
| 50                 | 16 | 283 | 43 | 10.6 | 258 | 36 | 9.0  | 1.11 | 0.16 | 0.040 |   |
| 60                 | 18 | 278 | 61 | 14.3 | 255 | 52 | 12.2 | 1.10 | 0.12 | 0.028 |   |
| 70                 | 17 | 276 | 51 | 12.4 | 260 | 32 | 7.8  | 1.06 | 0.11 | 0.006 |   |
| 80                 | 17 | 310 | 31 | 7.5  | 287 | 28 | 6.8  | 1.08 | 0.09 | 0.022 |   |
| 100                | 24 | 291 | 51 | 10.5 | 285 | 52 | 10.5 | 1.03 | 0.12 | 0.025 |   |
| 120                | 24 | 301 | 41 | 8.4  | 293 | 28 | 5.8  | 1.03 | 0.13 | 0.026 |   |
| mean normal values |    | 248 | 46 | 5.6  |     |    |      | 1.00 | 0.07 | 0.012 | C |
| 64 C parts         |    |     |    |      |     |    |      |      |      |       |   |

The radioactivity of the A parts (See Fig. 16 and Table) on the control side rose from normal values on the 10th day to higher than normal on the 40th day(\*) The activity then dropped to lower than normal on the 100th and 120th days(\*\*)

The activity of the A parts on the nailed side was higher than normal from the beginning of the experimental period(\*\*) and dropped through normal on the 60th day to lower than normal on the 100th and 120th days(\*\*)

The increase in activity on the control side in relation to the normal was about 25 % on the 30th day. On the nailed side the increase was about 45 % on the 10th day and 65 % on the 30th day. The activity at the end of the experiment was about 60–65 % of normal on both sides.

The activity in the A parts on the nailed side was higher than on the control side throughout the experimental period(\*\*\*) On the 10th day the activity on the nailed side was about 60 %, on the 30th day about 30 %, and at the end of the experiment about 10 % higher than on the control side.

The ash weight of the C parts (See Fig. 15 and Table) on the control side did not vary from normal from the 10th day to the 70th day. On the 80th day it had increased and stayed higher than normal during the rest of the experimental period(\*\*\*)

The ash weight of the C parts on the nailed side on the 10th day was higher than normal(\*) The ash weight then fell and on the 20th and 30th days it lay within the range of normal range of variation. Between the 30th and 40th days the ash weight increased again and during the rest of the experimental period it was higher than normal(\*\*)

On the control side the ash weight on the 80th day was about 15 % above normal and remained so throughout the rest of the experimental



period On the nailed side the ash weight on the 10th day was about 10 % on the 40th day about 20 % and on the 80th day about 25 % above normal This increase persisted largely unchanged until the 120th day

The ash weight of the C parts on the nailed side was higher than on the control side throughout most of the experimental period(\*) However on the 100th day and on the 120th day no difference in ash weight was found between the two sides

The activity of the C parts (See Fig. 16 and Table) on the control side showed a tendency to be above normal during the first half of the experimental period From about the 70th day the activity showed a falling tendency and on the 100th day it was lower than normal(\*\*)

The activity of the nailed C part on the 10th day was higher than normal(\*) and then increased to reach a maximum about the 40th day The activity then dropped and on the 80th day and during the rest of the experimental period it lay within the normal range of variation

The decrease in activity on the control side in relation to normal was about 20 % on the 100th day The increase in activity on the nailed side on the 10th day was about 50 % and on the 40th day it was about 90 %

The activity of the C parts on the nailed side was higher than on the control side throughout the experimental period(\*\*) The increase in activity in relation to the control side on the 10th day was about 30 % on the 40th day about 60 % and on the 120th day it was about 25 %

Estimation of mineral density of callus (Fig. 17 and Table) From day 10 the mineral density of the callus tended to increase on the nailed and on the unnailed side The increase was initially fairly abrupt but from the day 30 and during the rest of the experimental period it was more gentle The mineral density on the nailed side on day 10 was about 20 % greater(\*) and on day 20 about 40 % greater(\*\*\*) than on the control side During the rest of the experimental period there was no difference in the mineral density of the two sides

The mineral density on day 10 on the nailed side was about 45 % of normal against about 35 % on the control side On day 20 the mineral density rose to 55 % and 40 % respectively On day 60 the mineral density was about 70 % and on day 120 it was about 75 % of normal on both sides

Results of statistical analysis The qualitative analysis showed that apart from the 10 day group and for the nailed bones also the 20 day group during the first 50 days there was no covariation between the absolute tensile strength and the ash weight of the B parts the difference is seen

the observed and the calculated values for  $n_{12}$  being alternately positive and negative and of the same order as the mean error. After 50 days the difference was positive and, as a rule, larger and sometimes more than twice the mean error and thus showed a correlation.

Quantitative evaluation of the covariation between the absolute tensile strength and the ash weight of the B parts gave the following correlation coefficients (Table 3)

| age of fracture in days | correlation coefficients absolute tensile strength ashweight B parts |        |
|-------------------------|--|--------|
|                         | unnailed   | nailed |
| 10                      | 0.68   | 0.40   |
| 20                      | 0.01   | 0.44   |
| 30                      | -0.05  | -0.14  |
| 40                      | 0.23   | 0.03   |
| 50                      | -0.06  | -0.41  |
| 60                      | 0.50   | 0.61   |
| 70                      | 0.22   | 0.67   |
| 80                      | 0.38   | 0.54   |
| 100                     | 0.50   | 0.50   |
| 120                     | 0.44   | 0.52   |

Table 3

During the first 50 days, apart from the 10 day group and for the nailed bones also for the 20 day group there was no correlation between the absolute strength and the ash weight of the B parts. During this period when the absolute tensile strength increased rapidly while the ash weight decreased some other factors must have influenced the variation in the values found for the tensile strength among the experimental animals. After the 60th day on the other hand a correlation appeared which may broadly speaking be regarded as significant. Yet the relationship cannot be regarded as very strong since the coefficients of correlation were on the average about 0.40.

Qualitative analysis of the absolute tensile strength and the ash weight of the A parts, the activity of the B parts and the mineral density showed no relationship between these data.

## DISCUSSION

The shapes of the curves for the various measurements made during the course of healing of the fractures were principally the same for the nailed as for the unnailed bones. The first part of the discussion will therefore be confined to fracture healing as estimated from measurements of the control bones i.e. the unnailed bones, while the difference between the healing of the nailed and unnailed bones will be discussed in a separate section.

It is known from histological investigations that the healing of a fracture in the initial phase proceeds with great intensity (HERTZ 1936 URIST & McLEAN 1941 PRITCHARD & RUZICKA 1950). Multiple fractures do not appear to influence the course of healing (McLEAN & URIST 1950 BAUER 1952) and the calcification process of the callus closely resembles that in growing bone (McLEAN & URIST 1951). URIST & McLEAN (1941) found that the mass of the callus in the femur of the rat is maximal about the 10th—16th day and that the calcification of the callus is also greatest at that time (cf e.g. BAUER 1954c). The callus then decreases in amount and healing proceeds with reorganization of the callus and subsequent formation of the cortex.

The histological descriptions of fracture healing deal mainly with the early phase of healing and the literature on subsequent phases to definitive histological healing is scanty and contradictory. DOWN et al (1932) found that fractures of the fibula of the rat heal with histologically normal cortex within about 50 days while HERTZ (1936) still found remnants of necrotic cortical tissue in the new formed cortex (fibula fractures of guinea pigs) on the 50th day and he claimed that definitive healing requires a long time at least 1 year.

In the present investigation the biological reaction of bone to a fracture was studied by measuring the cross sectional area of the callus the ash weight and the radioactivity of the radius. Further repeated determination of the tensile strength of the callus was performed. It is clear from Figs 11, 13 and 14 that the curves for the area of the callus and for the ash weight and radioactivity of the fracture region resemble one another in shape. After an initial ascent the curves for the area and the activity reach a peak about the 20th day. The ash weight appears to reach

# CURVE DEMONSTRATING RISE AND FALL OF VASCULAR RESPONSE

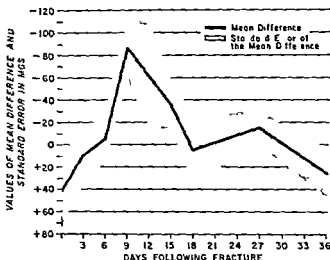


CHART I

Curve showing the rise and fall of the vascular response following fracture of the tibia in the rat. The values of the mean differences in milligrams are plotted against the number of days following fracture and are seen as a solid black line. The stippled band represents the range of the standard error of the mean differences.

Fig 18

Published with kind permission of The Journal of Bone and Joint Surgery

about the 30th day. Afterwards the curve first rapidly falls but from about the 50th—60th days the fall is lower, and at the end of the experimental period the values for the various measurements are still higher than normal.

WRAY & LYNCH (1959) determined the volume of the vascular bed in the hind legs of rats with fracture of the tibia. The curve in Fig 18 illustrates the course of the traumatic hyperaemia during the healing period of the fracture. WRAY & LYNCH found that the peak of the curve coincided in time with that of the maximum of callus. The chronological lag of the peak of the curve in relation to the present experiments may be due to differences in the sizes of the animal (cf. URIST & McLEAN 1941, BOHR & SORESEN 1950 and BAUER 1954c).

The curves in Figs 11, 13 and 14 show the initial rate of callus formation. When the callus reached its peak on about the 20th day the cross-sectional area was more than 4 times normal. The ash weight in the fracture area had increased by 80%, and the accretion rate was 8—9 times normal. The increase in ash weight is the net increase because during the initial phase of healing the ash weight of the fracture fragments decreases because of resorption. Judging from histological examination the resorption is greater

when the callus mass is maximal (URIST & McLEAN 1941) Micro-radiographical examinations by NILSSON (1959) however appear to show that the resorption in the fracture ends occurs earlier and reaches maximum at the same time as the commencing mineralization of the callus

The rapid fall in the curve between the 20th and 60th days for all three measurements shows the rate of reorganization of the callus mass During this period the area of the callus decreased by about 50 %, the ash weight by about 25 % and the rate of accretion by about 65 %. During the rest of the experimental period the curves fell slowly and even at the end of the experimental period the cross sectional area was still twice normal (see photo in appendix) while the ash weight had increased by about 20 % and the rate of accretion was more than 1.5 times normal

My findings agree with the histological appearance of the initial stage of healing of fractures and confirmed the results of BAUER (1954c) In his experiments on rats he found a similar increase in the ash weight and activity while COFF & GREENBERG (1945) and BOHR (1950) in their isotope experiments found a similar but smaller increase in activity over the region of the fracture

The results of the present investigation also showed that definitive healing of a fracture requires a long time and confirms the investigations of BAUER & CARLSSON (1955) BOHR (1955) and NILSSON (1959) and is in agreement with the histological findings of HERTZ (1936)

Measurements in man after the injection of  $\text{Sr}^{90}$  have shown a similar increase in radioactivity over the fracture (BAUER & WENDEBERG 1959) WENDEBERG (1961) in his material of tibia fractures found the activity over the fracture region to be up to 30 times higher than that over the intact bone The activity was found to be highest about 6—8 months after the fracture The rise in activity persisted for a long time WENDEBERG (1961) thus found an increased activity even in several year old fractures of the tibia

#### *Cross sectional area of marrow and cavities*

The restoration of the marrow cavity and the decrease in the total mass of callus appears to be related to the formation of the cavities observed in the callus The formation of such cavities has been described by BAST et al (1925) HERTZ (1936) and ESKELUND & PLTIN (1950) as a normal phase in the reorganization of callus The cavities are first seen in the periphery of the callus when the callus reaches its maximal size (HERTZ, ESKELUND & PLTIN) The cavities increase in size during the healing process and later they are scattered more widely in the callus (HERTZ) The cavities are

largest about 3—4 weeks after fracture (ESKELUND & PLUM fibula of rats), after which they decrease in size and number. The cavities contain osteogenetic tissue (BAST et al) and marrow tissue (ESKELUND & PLUM). They gradually decrease in size and number on formation of new bone and with simultaneous reduction in the amount of inner and outer callus (BAST et al).

The occurrence and the distribution of the cavities in the present material thus agree with these earlier observations (see Table I and photos in appendix).

### *The mineral density of callus*

While the total ash weight of the callus is increased during the healing of the fracture, the mineral content of the callus expressed by the ratio ash weight/B cross sectional area of callus is decreased. The use of the ratio between the ash weight in the region of the fracture and the cross sectional area of the callus as an expression of the density of the mineral in the callus can be justified on the following grounds. In the region of the B parts the normal rabbit radius is almost cylindrical in shape. When the amount of callus is greatest the B parts take on the shape of a double truncated cone. As healing proceeds they again become cylindrical. Since there exists a constant ratio between the volume of a cylinder and that of a cone and their respective bases (provided the height is constant) it appears reasonable to assume that there is also a largely constant ratio between the volume of the B parts and their cross-sectional area. Fig. 17 illustrates the course of the curves of the mineral density of the callus during the experimental period.

It is seen from the curve of the unnailed bones in Fig. 17 that the mineral density in the callus on the 10th day was about 35 % of normal. The curve then rose fairly sharply until about the 30th day, when the mineral density was about 60 %. At the same time the ash weight in the fracture region was 85 % above normal. The rise in mineral density then gradually increased and on the 120th day it was about 75 % of normal. The curve given for the mineral density in the callus (as expressed in the present investigation) corresponds in its course to that of the mineralization of the callus of the tibia of the rat and femur of the dog found by NILSSON (1959) on quantitative microradiography.

### *Changes in the A and C parts*

The effects caused by the fracture on the ends of the radius were most pronounced in the A parts (Figs 15 and 16) where the ash weight on about the 10th day was about 15% below normal. At the same time the activity showed a rising tendency but no deviation from normal value. It is clear from the course of the curve that the weight loss begins early during the first phase of healing and has passed its maximum already at the beginning of the observation period. At the same time the accretion does not deviate from normal.

The ash weight from the 10th day increased and reached normal values about the 30th day to remain within the normal range of variation until the 70th day. From about the 80th day the ash weight was significantly increased and remained increased until it returned towards normal at the end of the experimental period. The increase in ash weight from the 10th day was accompanied by a rise in the activity and about the 40th day it was increased. Then the activity fell and on about the 100th and 120th days it was significantly lower than normal.

The changes in the C-parts were correspondingly less pronounced. During the first half of the experimental period the ash weight and the activity lay within the normal range. On about the 70th day a rise occurred in the ash weight and at the same time the activity fell. The ash weight then persisted significantly increased throughout the rest of the experimental period while the activity around the 100th day was below normal and then again approached normal.

The changes in the activity and ash weight of the A parts during the first half of the experimental period agree largely with those found in earlier experiments by BAUER (1954c) and BAUER & CARLSSON (1955) but in the present experiments the changes were less pronounced and particularly of shorter duration. The reason why it was not possible to demonstrate similar changes in the C parts in the present investigation was presumably due to the C parts consisting mainly of cortical bone in which the mineral content as shown by BAUER (1954d) is not so readily resorbed as the mineral in the A parts representing the lower radius meta epiphysis.

The more pronounced changes in the ends of the fractured bones observed by BAUER (1954c) and BAUER & CARLSSON (1955) may be explained by the fact that these authors used small animals (rats) in which the general reaction of the skeleton is more pronounced than in larger animals (LACROIX 1956; NILSSON 1959).

WENDEBERG (1961) also found an increase in the activity over the knee and femur in human subjects with fracture of the tibia. In the present investigation the maximum of activity of the ends of the

somewhat later during the course of healing than in the region of the fracture. This could not be confirmed by WENDEBERG's investigation.

From about the 80th day an increase was noted in the ash weight and a decrease in the activity of both end parts of the bones. These changes persisted during the rest of the experimental period, although the ash weight of the A parts showed a tendency to a decrease about 120th day. In human beings with 4—5 year old fractures of the tibia WENDEBERG (1961) reported a similar decrease in the activity over the knee and femur on the fractured side where the activity was lower than that over the control knee and femur.

### *Tensile strength*

It is clear from Figs 9 and 10 that until the 80th day the tensile strength curves were almost linear. Then they became flatter, particularly the curve for the absolute tensile strength. The flattening coincided with the time of appearance of the defects in the radius (see Table 2), which in the 100 and 120 day groups occurred in one half to three fourths of the experimental bones. The decreased absolute tensile strength of the defective bones must therefore have a decisive effect on the shape of the curve and will influence the evaluation of the course of healing which is based on the tensile strength of normal intact radii.

In the curves for the absolute and specific strength (Figs 9 and 10) is also given — in thin lines — for the 70, 80, 100 and 120 day groups, the values corrected according to the calculation described on page 33. In view of the above observations, the tensile strength in relation to the normal tensile strength in the last mentioned experimental groups and particularly in the 100 and 120 day groups must be evaluated with caution.

It is however reasonable to suppose that the corrected shape of the curves which show an almost linear continuous ascent, is a more correct expression of the cumulative bone forming process occurring in the callus during healing of a fracture.

It is difficult to explain the development of such fusion between the marrow cavities of the two bones. One might imagine the defect to be due to a lesion of the ulna during the operation. But such a lesion is less likely because the ulna is protected by an osteotomy hook during operation. In addition it would be more reasonable to suppose that a lesion would heal instead of growing in size. Fusion of the radius and the ulna occurs so often that it seems to be a normal link in the healing process. A condition necessary for such fusion may be the close contact present between the two bones, a connection that is augmented still more by the fact that callus to some extent encroaches upon the ulna. But why the cortex at the site of osteo-



tion of the radius as well as the cortex of the ulna is resorbed with consequent communication between the marrow cavities of the two bones is still obscure

The shape of the tensile strength curve showed good agreement with results obtained by the other measurements of the course of healing of the fracture in the present investigation. These curves also showed that the healing process was not concluded at the end of the experimental period.

Good agreement was found between the present results and those described by HÄBLER & REISS (1936) on experiments in which they loaded healing fractures of the radius-ulna and tibia of the dog. When measuring the breaking strength of 50–90 day old fractures they found the absolute strength to be decreased to 70–80 % of normal.

COPP & GREENBERG (1945) carried out similar experiments on fractures of the fibula of the rat and found the absolute strength to be normal after about 30 days. These fractures appeared to heal rather rapidly. Simultaneous measurements of the radioactivity over the region of the fracture after administration of  $\text{Sr}^{85}$  showed an increased uptake suggesting that the healing process at that point was not concluded.

The total and the specific strength characterize the healing process in two different ways. The absolute strength gives the total strength of the callus while the specific strength is a measure of the quality of the callus tissue. A characteristic difference was found between the absolute and specific strength during the course of healing. While the absolute strength on the 10th day was about 4 % of normal the specific strength was only about 1 %. On the 30th day the corresponding values were 20 % and 6 % and on the 80th day 50 % and 20 %. It is thus apparent that fracture healing is achieved in the first place by a relative rapid increase in the absolute strength of the callus while the quality of the callus improves at a much slower rate.

In the treatment of fractures the time of clinical healing, i.e. when the callus is strong enough to permit weight bearing, is of greatest interest. PAUWELS (1946) stated that a bone is not normally loaded to more than about one seventh of what it can tolerate. If this be taken as a norm for clinical healing it will be found that the absolute strength of the fractured rabbit radius attains such strength by about the 20th–30th day, i.e. at the time when the amount of callus was largest and when the ash weight and the radioactivity in the region of the fracture were maximal. Thus clinical healing of a fracture occurs histologically and biologically in a relatively early stage.

This is in good agreement with the observation in my experiments that the animals soon moved about unhindered after the operation. These

observations also appear to be in accord with clinical experience. Fracture of the tibia in man will, as a rule, be clinically healed within 6—8 months: at the time when WENDEBERG (1961) found the radioactivity to be highest.

The shape of the curves for the absolute tensile and specific strength in the present experiments were largely linear and ascending. COPP & GREENBERG (1945) only studied the absolute strength in their experiments and found a similar rise. In identical experiments MCKEOWN et al (1932) found a deviating course for the strength curve which fell between the 15th and 20th days, coinciding with the time of restoration of the marrow cavity. These authors ascribed the fall in strength to the resorption of the endosteal callus. The decreasing strength in the experiments of MCKEOWN et al is difficult to explain but it could hardly be due to resorption of the endosteal callus because this process is a normal link in its reorganization.

*Ash weight, mineral density of the callus and radioactivity in relation to strength of callus*

HABLER & REISS (1936) found no relationship between the strength of the callus and the ash weight of the fracture region in dogs. BOHR (1955), on the other hand, was able to demonstrate such a relationship in his experiments from the 3rd week after the fracture.

In the present experiments a covariation was found between the absolute strength and the ash weight of the B parts from about day 60 and during the rest of the experimental period. No such covariation could be found — except on day 10 — during the first half of the experimental period. While the strength of intact cortical bone increases with the mineral content (BELL, CHAMBERS & DAWSON 1947; WIER, BELL & CHAMBERS 1949; VOSE & KUBALA 1959) this was not found to hold for the callus until the latter had reached a certain stage of maturity. Therefore from day 20 to day 60 other factors must be responsible for the increasing strength of the callus.

It is clear from Fig. 13 that the ash weight of the callus between the 30th and 60th days fell from 197 mg to 141 mg, i.e. by about 30 %. In the same interval the absolute strength rose from 18.5 kg to 38.2 kg, i.e. about 100 % (Fig. 9). Despite a considerable reduction in the ash weight there was a strong increase in the absolute strength of the callus. The cause of this rise should be sought in the reorganization of the callus occurring during the healing process.

The correlation found between the absolute tensile strength and the ash weight of the B parts on day 10 can be explained by the fact that the reorganization of the callus at this time is not yet so advanced as to exert any dominating effect on the healing of the fracture.

It is apparent from Fig 17 that the mineral density of the callus from day 30 to day 60 increased from about 60 % to 70 % of normal. During the same period the specific strength rose from 0.68 kg/mm<sup>2</sup> to 2.44 kg/mm<sup>2</sup> (Fig 10). At the same time as the mineral density increased by about 10 % the specific strength rose by about 250 %. The reorganization of the callus thus seems to be of importance also for the rapid increase of the specific strength.

In contrast to what was found by BOHR (1957) it was not possible to demonstrate any relationship between the absolute strength and the ash weight of the A parts — which consisted of the lower meta-epiphysis. This might be explained by the assumption that the variation in the degree of healing of the fractures in the different experimental groups in the present experiments was much smaller than in BOHR's experiments in which the fractures were not fixed. As in BOHR's experiments no correlation was found between the absolute strength and the radioactivity in the region of the fracture. This is explained by the fact that the activity measured does not give any information on the amount of bone formed but only on the rate at which new bone is formed.

#### *Discussion of healing of fracture in the nailed bones*

It is clear from Fig 11 that the callus formation in the nailed bones followed the same pattern as in the unnailed bones but that the cross sectional area on the 20th and 30th days was about 50 % and 25 % respectively less than in the unnailed bones. This finding is in agreement with observations made by previous investigators in experiments in which the amount of callus was judged radiographically (TRUEA & CAVALLAS 1955, FITTS Jr et al 1949, GALUZZI & GIANELLI 1953). All of these research workers also found the callus to form somewhat earlier in nailed bones. This could not be confirmed in the present experiments.

While the amount of endosteal callus in unnailed bones is rapidly decreased by resorption in nailed bones (Fig 12) the amount of endosteal callus as judged from the cross sectional area of the marrow (+ nail cavity) remained unchanged from the beginning of the experimental period until about the 60th day. Not until after the 60th day did the area of the marrow increase to reach normal value about the 70th day. The reorganization of the endosteal callus is thus retarded in the nailed fractures. This is also illustrated by the cavity formation. The cavities formed later and persisted longer in the nailed bones (See Table 1 and photographs in appendix). Delayed resorption of the endosteal callus of nailed bones has also been observed by GREISSMANN & REICH (1944) in experiments on dogs.

Also the formation of callus, as measured by the ash weight and the radioactivity in the B parts (Figs 13 and 14) was fundamentally the same for the nailed and unnailed bones, but both the ash weight and the activity (apart from the activity on the 10th day) during the initial active phase were less than the values noted for the control bones. After the 100th day the ash weight of the nailed fractures showed a tendency to increase, and on the 120th day it was significantly larger than that of the control bones. The activity after the 70th day accordingly showed a tendency to exceed that of the control bones, and on the 80th and 120th days it was higher than on the control side (Fig. 14).

Thus in the last part of the experimental period the values found for ash weight and radioactivity of the nailed bones showed a tendency to exceed those of the control bones. This might reasonably in part be ascribed to the delayed reorganization of the endosteal callus.

In the end parts of the nailed bones the changes in ash weight and activity were more pronounced than in the control bones. The shape of the curves showing the changes in the end parts (see Figs 15 and 16) were roughly parallel on both sides, which indicates that there was only a difference in degree of the changes.

The nail produced an increased accretion of bone salt in the ends of the radius from the very beginning of the experiment. This is in agreement with the histological changes observed in the periosteum, the cortex and the endosteum of nailed experimental fractures. Thus GRIESSMANN & REICH (1944), RAISCH (1943-44) and TRUETA & CAVADIAS (1955) observed the formation of periosteal new bone along the major part of the diaphysis in their experiments. This could not be observed on gross inspection of the experimental preparations in the present investigation even though the callus in the nailed bones showed a tendency to extend further along the diaphysis. This can probably be ascribed to the fact that in the present experiments the animals were practically full grown and as shown by TRUETA & CAVADIAS (1955) for example such animals show a less pronounced tendency to periosteal reaction than younger animals. TRUETA and CAVADIAS (1955) found that in unnailed fractured bones endosteal callus formed only in the proximity of the fracture. Similar observations were made in the present investigation. In nailed bones on the other hand more or less continuous new formed endosteal bone was seen along the entire length of the nail (GRIESSMANN & REICH 1944, RAISCH 1943-44). Similar changes have been described in human nailed fractures (ROTH 1945). In the present investigation such new formed callus was also observed and was most pronounced on the radial side of the radius. The changes in the cortex proved to be necrosis in the inner part of the latter (TRUETA &

CAYADIAS 1955) of signs of reorganization in the cortex (GRIFSBOMAN & REICH 1944 HASCHE KLUNDAR 1952) The reorganization of the cortex is a slow process TRUETA & CAYADIAS (1955) found traces of necrosis in the cortex 6—8 months after the fracture

The changes in the A and C parts of the nailed bones like the changes in the callus during the first part of the experimental period were characterized by a biphasic course The initial phase with increasing activity and ash weight was followed by a decrease in both indicating advancing reorganization of the new formed bone tissue in the endosteum periosteum and cortex During the latter part of the experimental period the activity began to fall as in the control groups while the ash weight increased

The values found for the absolute and specific tensile strength of the nailed fractures showed the same linear course found for the control side (Figs 9 and 10) The cortical defects in the nailed bones began to occur at the same time as in the nailed bone and to the same extent (see Table 2) What was said previously about the significance of these defects for the absolute and specific tensile strength and for the determination of the area of the callus and marrow also holds for the nailed fractures

The specific tensile strength found for nailed fractures between the 10th and 30th days was more than 50 % greater than that of the control fractures No difference in absolute tensile strength was found between the two sides during this interval From about the 40th day both the absolute and specific tensile strength of the nailed bone showed a tendency to be less than that of the unnailed bone and between the 50th and 70th days both the absolute and specific tensile strength were greater on the unnailed side About the 60th day the absolute strength was about 30 % and the specific strength about 37 % greater

The mineral density of the callus (given as ratio between the ash weight B and the area of the callus) between the 10th and 30th day was about 40 % greater on the nailed side than on the control side The specific tensile strength as well as the mineral density of the callus expressed in the ratio given above is measure of the callus quality Both showed that during the interval between the 10th and 30th days the callus was of better quality on the nailed side The higher density of the callus found in the nailed fractures agreed with the finding of TRUETA & CAYADIAS (1955) and FITTS Jr et al (1949) who radiographically observed a more advanced filling of the fracture space on the nailed side No difference was found in the absolute tensile strength between the callus on either side This can be explained by the fact that about the 20th day the cross sectional area was about 50 % greater on the unnailed than on the nailed side

In the interval between the 50th and 70th days there was no significant difference in the mineral density of the callus on either side and no difference was found between the cross sectional areas of the callus. Nevertheless the absolute and specific tensile strength of the unnailed bones were found to be greater. This difference in strength coincided in time with a marked reduction of the endosteal callus in the nailed bone (see Fig 12), and it may be ascribed in part to the delayed reorganization of the latter. After the 70th day no difference was found, but the absolute strength of the nailed bone tended to be less than that of the unnailed bone throughout the rest of the experimental period. This tendency was not so pronounced for the specific tensile strength however. The difference in strength was so great and the endosteal callus represents such a small portion of the total mass of callus that it is reasonable to assume, that a delayed reorganization in the endosteal callus alone cannot by itself explain the differences in strength.

If the reduction in the amount of callus is taken as a measure of the rate at which the callus is reorganized, the reduction in the mass of callus on the unnailed side between the 20th and 60th days was about 55 % while the corresponding reduction on the nailed side was about 35 %. It therefore appears reasonable to assume that a delayed reorganization of the callus on the nailed side not only occurred in the endosteal callus but also in other components of the callus mass.

Since the fractures were identical it would appear that the difference in the amount of callus and in tensile strength between the nailed and the unnailed bones were due to the nail *per se*. The actual mechanism involved is obscure.

According to KUNTSCHER the nail serves not only to give good fixation of the fracture but also mechanically stimulates fracture callus formation so that the callus formed is more abundant (KUNTSCHER 1955, 1958). The results of experimental nailing showed however that the fracture callus formed in less amount (FITTS Jr et al 1949, GAIUZZI & GIANFILI 1953, TRUETA & CAVADIAS 1955). This was confirmed by the present investigation. It may thus be excluded that the nail has any stimulating effect on the amount of callus formed and some authors, such as FITTS Jr et al (1949), GRIESSMANN & REICH (1944), RAISCH (1943-44) also believed that the effect on the callus formation should be ascribed entirely to the good fixation of the fracture secured by the nail.

KUNTSCHER (1958) does not believe the effect of the nail on the marrow circulation to have any appreciable influence on fracture healing. TRUETA & CAVADIAS (1955) on the other hand feels that the injuries to the marrow vessels by the nail can explain its effect on the callus formation.

However the vascular lesions alone do not seem to be able to explain

the effect of the nail. In fracture callus in nailed bones there is histologically a regularly radial arrangement of new formed bone trabeculae while in the callus of the control fractures the trabeculae are arranged irregularly (KUNTSCHER 1957, RAISCH 1943—44, GRIESSMANN & REICH 1944). Experiments have also shown that in contrast to unnailed bone the ossification in nailed bones occurs via pure fibrous callus without any intermediate cartilaginous phase (TRUETA & CAVADIAS 1955, RAISCH 1943—44, GRIESSMANN & REICH 1944). This course in the ossification cannot however be ascribed to any specific effect of the nail. A similar callus formation has been observed in unnailed fracture with ideal fixation (KAPSHAMMER 1898, MATZEN 1954) while even in nailed fractures with poor fixation cartilaginous callus formation has been reported (COURTOIS SUFFIT 1952, GRANJON & SOEUR 1955).

According to PRITCHARD & RUZICKA (1950) the course of the ossification of the callus is determined by the vascularization of the latter. Cartilaginous callus forms when vascularization is poor. PRITCHARD & RUZICKA claim that the significance of fracture fixation consists mainly in securing good conditions for vascularisation of the callus. It would therefore appear reasonable to suppose that the higher specific tensile strength found in nailed bones on about the 20th day was due mainly to the better fracture fixation secured by the nail. The smaller amount of callus found at that time in the nailed bones might also be explained by better fracture fixation. For clinical experience has shown that the amount of callus formed varies with the degree of fixation of the fracture.

The lower absolute and specific tensile strength found for the nailed bones on about the 60th day may most likely be ascribed to the lower reorganization observed in the callus. That this slower reorganization should be related to vascular changes caused by the nail appears less likely because TRUETA & CAVADIAS (1955) found the vascularization of the callus in the nailed radius of the rabbit to be the same as for the control fractures. No satisfactory explanation can be offered for the delay but it might possibly be due to mechanical factors. PAUWELS (1946) for example claims that just as the shape and structure of bones is to a certain extent determined by mechanical forces (Wolff's law) the reorganization of the callus is due *inter alia* to the bending and traction forces which influence the callus during the healing process. In accordance with this it would not be unreasonable to imagine that the nail by its presence prevents or reduces the effect of the external forces and that the reorganization of the callus in nailed bones therefore proceeds at a slower rate.

As mentioned, when loaded in the traction apparatus 4 of the bones (3 in the 100 day group and 1 in the 120 day group) fractured outside the

site of osteotomy. All of these bones had been nailed and healed without defects. Though these findings do not allow of any valid conclusions, they are of interest in the light of the necrosis demonstrated by others in the inner cortex of the diaphysis of nailed bones. It cannot be excluded that this might reduce the strength of the cortex outside the site of the fracture during reorganization of the necrotic tissue. The decrease in the tensile strength compared with normal was on the average 30—35 %. Owing to the enormous strength of the bone in relation to the strain and stress to which it is normally subjected (PAUWELS 1946) this reduction in the tensile strength is of theoretic interest rather than of practical importance.

The decrease in the total strength of the nailed bones between the 50th and 70th days was about 25 % in relation to that of the unnailed bones. At first glance this difference seems considerable. Clinically, it appears in a relatively late stage of fracture healing and is only of temporary nature. With reference to what was said previously about clinical healing and the order of the physiological load, the decrease in the total strength of the callus cannot be of any appreciable importance.

The possible effect of early extraction of the nail on the decrease in the tensile strength was not studied in the present investigation. To achieve any such effect, the nail should be extracted as early as possible, i.e. about the 20th to 30th days, when the fracture is clinically healed.

In clinical practice the time of extraction is dictated by the radiographical appearance of the fracture and clinical experience. LARITZEN (1949) extracts nails from humerus fractures after 3—5 months and from tibia fractures after 6—12 months. BOHLER (1948) gave 6—7 months as a suitable time for extraction of nails. Clinical healing of a fracture, as shown in the present investigation, occurs fairly early in the course of healing and the extraction, as shown above, is done so early that it should be possible to avoid or reduce the undesirable effect of the nail.



## SUMMARY

The course of healing of unnailed and identical nailed fractures in rabbits was studied from the 10th to the 120th day following open osteotomy of the radius on both sides. The absolute tensile strength of the callus was determined in a traction apparatus. The transverse area of the callus at the site of osteotomy was measured by planimetry of reproductions on photographic paper. Hence the specific tensile strength of the callus was calculated. Most of the animals were given  $\text{Sr}^{85}$  4 days before they were killed. Determinations were made of the ash weights and of the radioactivity in the region of the fracture and in the ends of the bone. The ratio between the ash weight in the region of the fracture and the transverse area of the callus was calculated to give the mineral density of the callus.

The following observations were made:

1. Fixation of the fracture on the nailed side was better than on the contralateral side.

2. During the course of healing gross cavities of different shape, size and number developed in the nailed and in the unnailed bone. The cavities appeared later and persisted longer in the nailed bones.

3. During the last third of the experimental period both the radius and the ulna in an increasing number of animals showed a cortical defect at the level of the osteotomy. At this level a connection arose between the marrow cavities of the two bones.

4. The periosteal callus tended to spread longer along the diaphysis on the nailed side than on the unnailed side. On the unnailed side endosteal callus formed only in the region of the osteotomy, but more or less continuously along the entire length of the nail on the other side.

5. The absolute as well as the specific tensile strength of the nailed and of the unnailed bones showed an almost even increase throughout most of the experimental period, but the increase of the absolute tensile strength was more rapid. While the absolute tensile strength of the unnailed bone was about 1% of normal on the 10th day, about 20% on the 30th day, and 50% of normal on the 80th day, the corresponding values for the specific tensile strength were 1%, 6% and 20% respectively. The specific tensile strength on the nailed side was greatest about the 20th day, while the absolute tensile strength at that time was the same on both sides. After

the 40th day the absolute as well as the specific tensile strength of the unnailed bones showed a tendency to increase more than that of the nailed bones, and the absolute as well as the specific tensile strength of the unnailed bones was greatest about the 60th day. This difference afterwards disappeared.

6 The biological reaction of the bone in the region of the fracture, as reflected in the transverse area of the callus, the ash weight and the activity, showed largely the same course. After an initial increase with a maximum about the 20th day (for ash weight about the 30th day) a fall occurred, which was initially steep. From about the 50th to 60th day the fall was slower, but at the end of the experimental period the transverse area as well as the ash weight and the activity were still increased in relation to normal.

During the initial active phase the transverse area of the callus, the ash weight and the activity were greater on the unnailed side. When the callus was maximal the transverse area of the unnailed side was about 4 times and on the nailed side about 3 times greater than normal, the ash weight was increased by about 85 % and 65 %, respectively, and the activity was 8.3 and 5.8 times respectively greater than normal. No difference was found between the two sides after the 40th—50th day. At the end of the experimental period the transverse area of the radius on both sides was twice normal, the ash weight was increased by about 25 %, and the activity was almost twice normal. The reorganization processes in the callus as measured by the variation in the transverse area of the callus, the ash weight and the activity in the region of the fracture thus showed a more rapid course on the unnailed side.

The amount of endosteal callus, as measured by the area of the bone marrow, showed largely the same variation as the total mass of callus. On the 20th day the marrow cavity on the unnailed side was filled with callus. On the nailed side the amount of endosteal callus was less. While the amount of endosteal callus on the unnailed side decreased rapidly so that the marrow cavity on the 60th day was of normal size, the amount of endosteal callus on the nailed side was unchanged in amount from the beginning of the experimental period until the 60th day. Not until the 70th day was the marrow cavity of normal size. At the end of the experimental period the marrow cavity was larger than normal on the unnailed side while on the nailed side it tended to be larger.

7 The mineral density of the callus on the nailed and on the unnailed side increased from the beginning of the experimental period. The increase was initially relatively rapid but afterwards slower. On the 20th day it was about 55 % of normal on the nailed side and about 40 % of normal on the

unnailed side afterward, no difference in mineral density was found between the two sides. At the end of the experimental period the mineral density of the callus was about 75 % of normal.

8 The end parts of the radius on the unnailed side showed changes which were most pronounced in the distal metaphyses. At the beginning of the experimental period the ash weight on the unnailed side had decreased to about 85 % of normal and at the same time the activity showed no deviation from normal. After an increase to normal the ash weight increased further and from the 80th day it was above normal while after an increase to above normal the activity again fell and from the 80th day it was below normal. During the latter part of the experimental period similar changes were demonstrated in the proximal end part of the radius while no variation from normal was found in that part during the first two thirds of the investigation period.

The changes in the ash weight and activity of the end parts of the nailed and unnailed bones were parallel throughout the experimental period but both the ash weight and the activity were greater on the unnailed side.

9 From the 60th day and during the rest of the experimental period both the unnailed and the nailed bones showed a correlation between the absolute tensile strength and the ash weight of the fracture region. In the first half of the experimental period no such correlation was found for the unnailed side (apart from the 10th day) or the nailed side (apart from the 10th and 20th days).

These observations suggest the following conclusions:

1 During healing of a fracture the absolute tensile strength of the callus increases more rapidly than the specific tensile strength.

2 Definitive healing of a fracture requires a long time.

3 Changes in the mineral metabolism are not confined to the region of the fracture.

4 Not until a relatively late stage of healing does a correlation appear between the absolute tensile strength of the callus and the ash weight in the region of the fracture resembling that seen in intact cortical bone.

5 The course of healing is largely the same in nailed and unnailed bones though nailing causes an increased mineral metabolism in the end parts of the nailed bones.

6 The amount of callus formed is less on the nailed side.

7 In the initial stage of healing the specific tensile strength and the mineral density of the callus are greater on the nailed side while the absolute tensile strength is the same on both sides.

8 In the later stage of healing the absolute and the specific tensile strength are greater on the unnailed side.

Table 1 *Summary of material*

| age of fracture in days | No of animals | wt at op in kg |     |               | wt at death in kg |     |               | length of unnailed radius in cm |     |               | length of nailed radius in cm |     |               |
|-------------------------|---------------|----------------|-----|---------------|-------------------|-----|---------------|---------------------------------|-----|---------------|-------------------------------|-----|---------------|
|                         |               | mean           | s d | error of mean | mean              | s d | error of mean | mean                            | s d | error of mean | mean                          | s d | error of mean |
| 10                      | 24            | 2.4            | 0.2 | 0.04          | 2.3               | 0.2 | 0.03          | 6.6                             | 0.2 | 0.05          | 6.6                           | 0.2 | 0.05          |
| 20                      | 28            | 2.5            | 0.3 | 0.06          | 2.3               | 0.3 | 0.06          | 6.5                             | 0.3 | 0.06          | 6.5                           | 0.3 | 0.06          |
| 30                      | 24            | 2.5            | 0.3 | 0.05          | 2.7               | 0.3 | 0.05          | 6.7                             | 0.2 | 0.05          | 6.7                           | 0.2 | 0.05          |
| 40                      | 24            | 2.5            | 0.4 | 0.08          | 2.5               | 0.3 | 0.07          | 6.6                             | 0.2 | 0.04          | 6.6                           | 0.2 | 0.04          |
| 50                      | 24            | 2.3            | 0.1 | 0.03          | 2.4               | 0.2 | 0.04          | 6.6                             | 0.2 | 0.04          | 6.6                           | 0.2 | 0.04          |
| 60                      | 24            | 2.2            | 0.1 | 0.03          | 2.4               | 0.3 | 0.05          | 6.6                             | 0.2 | 0.05          | 6.6                           | 0.2 | 0.05          |
| 70                      | 24            | 2.3            | 0.1 | 0.03          | 2.5               | 0.3 | 0.05          | 6.7                             | 0.2 | 0.04          | 6.7                           | 0.2 | 0.05          |
| 80                      | 24            | 2.4            | 0.2 | 0.03          | 2.6               | 0.2 | 0.03          | 6.7                             | 0.2 | 0.05          | 6.7                           | 0.2 | 0.04          |
| 100                     | 24            | 2.2            | 0.1 | 0.03          | 2.6               | 0.3 | 0.06          | 6.8                             | 0.2 | 0.03          | 6.7                           | 0.2 | 0.04          |
| 120                     | 24            | 2.2            | 0.2 | 0.03          | 2.6               | 0.3 | 0.06          | 6.8                             | 0.2 | 0.05          | 6.8                           | 0.2 | 0.05          |
| mean normal values      | 32            | 2.5            | 0.2 | 0.03          |                   |     |               | 6.6                             | 0.2 | 0.04          | 6.6                           | 0.2 | 0.04          |

| age of fracture in days | No of animals | distance of fracture from distal end of radius in cm unnailed |     |               | distance of fracture from distal end of radius in cm nailed |     |               | cross section of nail in mm <sup>2</sup> |     |               | length of nail in cm |     |               |
|-------------------------|---------------|---|-----|---------------|---|-----|---------------|--|-----|---------------|----------------------|-----|---------------|
|                         |               | mean  | s d | error of mean | mean  | s d | error of mean | mean                                     | s d | error of mean | mean                 | s d | error of mean |
| 10                      | 24            | 3.1   | 0.5 | 0.10          | 3.1   | 0.4 | 0.08          | 1.1                                      | 0.3 | 0.07          | 5.1                  | 0.6 | 0.12          |
| 20                      | 28            | 3.2   | 0.5 | 0.09          | 3.2   | 0.6 | 0.10          | 1.6                                      | 0.3 | 0.05          | 5.1                  | 0.8 | 0.15          |
| 30                      | 24            | 3.3   | 0.4 | 0.08          | 3.3   | 0.4 | 0.08          | 1.4                                      | 0.3 | 0.06          | 5.5                  | 0.9 | 0.17          |
| 40                      | 24            | 3.2   | 0.5 | 0.10          | 3.2   | 0.5 | 0.11          | 1.5                                      | 0.3 | 0.05          | 5.1                  | 0.7 | 0.14          |
| 50                      | 24            | 3.0   | 0.3 | 0.05          | 3.0   | 0.3 | 0.06          | 1.5                                      | 0.3 | 0.07          | 5.0                  | 0.4 | 0.08          |
| 60                      | 24            | 3.0   | 0.3 | 0.05          | 3.0   | 0.3 | 0.05          | 1.4                                      | 0.3 | 0.07          | 4.9                  | 0.5 | 0.09          |
| 70                      | 24            | 3.1   | 0.3 | 0.05          | 3.0   | 0.3 | 0.06          | 1.3                                      | 0.3 | 0.05          | 5.0                  | 0.6 | 0.12          |
| 80                      | 24            | 3.1   | 0.3 | 0.06          | 3.0   | 0.3 | 0.06          | 1.5                                      | 0.3 | 0.06          | 5.0                  | 0.5 | 0.10          |
| 100                     | 24            | 3.1   | 0.2 | 0.05          | 3.1   | 0.2 | 0.05          | 1.7                                      | 0.2 | 0.04          | 4.8                  | 0.3 | 0.06          |
| 120                     | 24            | 3.1   | 0.2 | 0.05          | 3.1   | 0.2 | 0.05          | 1.5                                      | 0.4 | 0.07          | 4.8                  | 0.3 | 0.06          |

Table 2 A Mean normal values

| animals used for determination of normal values for strength |          |     |               |             |                        |     |               |  |      |               |  |      |               |
|--|----------|-----|---------------|-------------|------------------------|-----|---------------|--|------|---------------|--|------|---------------|
| No of animals  | wt in kg |     |               | No of bones | length of radius in cm |     |               | distance of fracture from distal end of radius in cm |      |               | cross section of marrow in mm <sup>2</sup> |      |               |
|  | mean     | s d | error of mean |             | mean                   | s d | error of mean | mean   | s d  | error of mean | mean                                       | s d  | error of mean |
| 16   | 2.5      | 0.1 | 0.03          | 16 right    | 6.6                    | 0.2 | 0.05          | 3.1  | 1.0  | 0.9           | 3.1  | 1.3  | 0.11          |
|  |          |     |               | 16 left     | 6.6                    | 0.2 | 0.05          | 3.1  | 0.8  | 0.21          | 2.6  | 0.9  | 0.24          |
|  |          |     |               | 32(r+1)     |                        |     | 3.1           | 0.9  | 0.16 | 2.9           | 1.2  | 0.91 |               |

Continued

| animals used for determination of normal values for strength |          |     |               |             |  |     |               |                                 |     |               |   |      |               |
|--|----------|-----|---------------|-------------|--|-----|---------------|---------------------------------|-----|---------------|---|------|---------------|
| No of animals  | wt in kg |     |               | No of bones | cross section of bone in mm <sup>2</sup> |     |               | absolute tensile strength in kg |     |               | specific tensile strength in kg/mm <sup>2</sup> |      |               |
|  | mean     | s d | error of mean |             | mean                                     | s d | error of mean | mean                            | s d | error of mean | mean  | s d  | error of mean |
| 16   | 9.5      | 0.1 | 0.03          | 16 right    | 7.7                                      | 0.4 | 0.17          | 844                             | 130 | 3.26          | 11.01   | 1.55 | 0.387         |
|  |          |     |               | 16 left     | 7.51                                     | 1.0 | 0.2           | 839                             | 139 | 3.48          | 11.301  | 1.45 | 0.376         |
|  |          |     |               | 32(r + l)   | 7.6                                      | 0.8 | 0.15          | 842                             | 133 | 2.35          | 11.16   | 1.4  | 0.272         |

1) mean value for 15 bones only one bone splintered and therefore excluded

Table 2 B Mean normal values

Continued

| animals used for determination of normal values for cross section of bone and marrow |          |     |                                |                        |     |               |  |     |               |
|--|----------|-----|--------------------------------|------------------------|-----|---------------|--|-----|---------------|
| No of animals  | wt in kg |     | No of bones                    | length of radius in cm |     |               | cross section of marrow in mm <sup>2</sup> |     |               |
|  | mean     | s d |                                | mean                   | s d | error of mean | mean                                       | s d | error of mean |
| 16   | 2.4      | 0.2 | 16 right<br>16 left<br>32(r+1) | 6.6                    | 0.2 | 0.06          | 3.2  | 0.9 | 0.22          |
|  |          |     |                                | 6.6                    | 0.2 | 0.06          | 3.3  | 0.9 | 0.23          |
|  |          |     |                                |                        |     |               | 3.3  | 0.9 | 0.16          |
|  |          |     |                                |                        |     |               | 8.5  | 1.2 | 0.29          |
|  |          |     |                                |                        |     |               | 8.6  | 1.2 | 0.31          |
|  |          |     |                                |                        |     |               | 8.6  | 1.2 | 0.21          |

Continued

| animals used for determination of normal values of ash weight and activity |                      |     |               |        |     |               |  |     |               |        |     |               |        |     |               |        |     |               |     |     |      |
|--|----------------------|-----|---------------|--------|-----|---------------|--|-----|---------------|--------|-----|---------------|--------|-----|---------------|--------|-----|---------------|-----|-----|------|
| Sr <sup>85</sup> activity in % of dose $\times 10^2$                       |                      |     |               |        |     |               |  |     |               |        |     |               |        |     |               |        |     |               |     |     |      |
| No of bones  | wt of bone ash in mg |     |               |        |     |               | Sr <sup>85</sup> activity in % of dose $\times 10^2$ |     |               |        |     |               |        |     |               |        |     |               |     |     |      |
|  | A part               |     |               | B part |     |               | C-part   |     |               | A part |     |               | B part |     |               | C-part |     |               |     |     |      |
|  | mean                 | s d | error of mean | mean   | s d | error of mean | mean   | s d | error of mean | mean   | s d | error of mean | mean   | s d | error of mean | mean   | s d | error of mean |     |     |      |
| 32 r   | 159                  | 22  | 3.9           | 105    | 13  | 2.3           | 218  | 46  | 8.0           | 218    | 46  | 8.0           | 11.3   | 5.9 | 1.04          | 2.2    | 0.9 | 0.15          | 5.1 | 2.0 | 0.35 |
| 32 l   | 161                  | 21  | 3.8           | 108    | 15  | 2.6           | 218  | 46  | 8.1           | 218    | 46  | 8.1           | 11.4   | 5.9 | 1.04          | 2.2    | 0.8 | 0.15          | 5.1 | 2.0 | 0.35 |
| 64(r + l)  | 160                  | 21  | 2.8           | 107    | 14  | 1.8           | 218  | 45  | 5.6           | 218    | 45  | 5.6           | 11.4   | 5.8 | 0.73          | 2.2    | 0.8 | 0.10          | 5.1 | 2.0 | 0.24 |

Table 3 Values noted for 10 day old fractures

| animal<br>No     | cross section of marrow<br>in mm <sup>2</sup> |        | cross section of callus<br>in mm <sup>2</sup> |        | absolute tenile strength<br>in kg |        | specific tensile strength<br>in kg/mm <sup>2</sup> |        |
|------------------|---|--------|---|--------|-----------------------------------|--------|--|--------|
|                  | unnailed                                      | nailed | unnailed                                      | nailed | unnailed                          | nailed | unnailed   | nailed |
| 103              | 0   | 2.9    | 23.1  | 16.8   | 2.3                               | 1.2    | 0.10   | 0.07   |
| 104              | 0   | 2.5    | 20.5  | 16.6   | 3.5                               | 3.5    | 0.17   | 0.21   |
| 105              | 0   | 3.1    | 26.5  | 33.4   | 2.0                               | 3.2    | 0.08   | 0.10   |
| 106              | 0   | 2.5    | 24  | 21.6   | 4.7                               | 2.7    | 0.11   | 0.13   |
| 110              | 0   | 2.6    | 22.3  | 19.0   | 1.8                               | —      | 0.08   | —      |
| 111              | 2.4   | 2.7    | 39.8  | 26.5   | 3.7                               | 2.7    | 0.09   | 0.10   |
| 112              | 0   | 1.8    | 18.6  | 21.8   | 2.3                               | 1.8    | 0.12   | 0.08   |
| 113              | 0.4   | 4.1    | 25.0  | 19.4   | 3.7                               | —      | 0.15   | —      |
| 160              | 0   | 2.6    | 20.7  | 22.5   | 3.5                               | 3.2    | 0.17   | 0.14   |
| 161              | 0   | 2.6    | 23.1  | 22.7   | 3.2                               | 1.0    | 0.14   | 0.18   |
| 162              | 0   | 2.6    | 26.1  | 17.1   | 4.0                               | 4.9    | 0.15   | 0.21   |
| 163              | 0   | 1.0    | 33.3  | 26.6   | 5.5                               | 4.7    | 0.17   | 0.16   |
| 240              | 0   | 2.1    | 5.8   | 21.7   | 2.2                               | 1.0    | 0.03   | 0.20   |
| 241              | 0   | 3.7    | 33.0  | 3.8    | 3.0                               | 2.5    | 0.09   | 0.07   |
| 242              | 3.3   | 3.2    | 21.4  | 17.7   | 2.2                               | 2.0    | 0.09   | 0.11   |
| 243              | 0   | 2.2    | 30.8  | 35.1   | 2.3                               | 2.5    | 0.08   | 0.07   |
| 244              | 0   | 2.1    | 31.3  | 18.2   | 3.5                               | 3.2    | 0.11   | 0.18   |
| 34               | 0.4   | 0.5    | 24.6  | 24.8   | 1.2                               | 2.7    | 0.17   | 0.10   |
| 33               | 0   | 0.5    | 20.2  | 30.0   | 4.3                               | 3.5    | 0.11   | 0.12   |
| 34               | 0.1   | 1.6    | 21.5  | 11.4   | 4.5                               | 4.5    | 0.21   | 0.27   |
| 35               | 2.1   | 3.0    | 18.8  | 15.1   | 2.0                               | 2.0    | 0.11   | 0.11   |
| 36               | 0.7   | 1.4    | 25.8  | 40.2   | 4.2                               | 2.3    | 0.16   | 0.11   |
| 37               | 0.3   | 1.1    | 13.6  | 14.9   | 1.7                               | 3.0    | 0.07   | 0.09   |
| 38               | 0   | 0.6    | 26.1  | 16.9   | 3                                 | 4.2    | 0.13   | 0.16   |
| mean             | 0.4   | 2.2    | 25.0  | 22.8   | 3.2                               | 3.1    | 0.13   | 0.14   |
| s.d.             | 0.9   | 1.0    | 5.6   | 6.3    | 1.0                               | 1.0    | 0.05   | 0.06   |
| error<br>of mean | 0.18  | 0.20   | 1.15  | 1.28   | 0.20                              | 0.21   | 0.010  | 0.012  |

fractured during preparation of specimen

Table 4 Values not  $t$  day old fractures

| animal No       | cross section of marrow in mm <sup>2</sup> |       | cross section of callus in mm <sup>2</sup> |       | absolute tensile strength in kg |       | specific tensile strength in kg/mm <sup>2</sup> |                |
|-----------------|--|-------|--|-------|---------------------------------|-------|---|----------------|
|                 | unnailed                                   | naild | unnailed                                   | naild | unnailed                        | naild | unnailed  | naild          |
| 114             | 0  | 23    | 36.2                                       | 19.8  | 67                              | 9.5   | 0.19  | 0.48           |
| 121             | 0  | 7.5   | 23.5                                       | 26.8  | —                               | 7.2   | — <sup>1</sup>                                  | 0.27           |
| 122             | 0  | 11    | 42.1                                       | 25.9  | 7.5                             | 3.5   | 0.18  | 0.14           |
| 123             | 0  | 3.6   | 36.5                                       | 29.2  | 7.0                             | 8.5   | 0.19  | 0.29           |
| 15 <sup>a</sup> | 0  | 4.1   | 32.5                                       | 23.2  | 7.2                             | 8.8   | 0.22  | 0.38           |
| 153             | 0  | 4.1   | 46.4                                       | 25.8  | 7.2                             | 9.8   | 0.16  | 0.38           |
| 154             | 0  | 1.5   | 32.6                                       | 18.8  | 7.2                             | 9.8   | 0.22  | 0.52           |
| 155             | 0  | 4.6   | 34.4                                       | 20.4  | 8.8                             | 7.2   | 0.26  | 0.35           |
| 204             | 0  | 4.3   | 31.8                                       | 19.3  | 5.4                             | —     | 0.17  | — <sup>1</sup> |
| 205             | 0  | 1.4   | 38.2                                       | 25.6  | 14.2                            | 12.4  | 0.37  | 0.48           |
| 230             | 0  | 2.3   | 49.9                                       | 40.3  | 8.0                             | 15.0  | 0.16  | 0.37           |
| 231             | 0  | 2.9   | 42.7                                       | 35.2  | 18.6                            | 16.8  | 0.44  | 0.67           |
| 232             | 0  | 1.5   | 39.5                                       | 22.8  | 5.4                             | 10.6  | 0.14  | 0.46           |
| 233             | 0  | 1.6   | 35.4                                       | 30.2  | 8.0                             | 5.4   | 0.23  | 0.18           |
| 234             | 0  | 2.1   | 45.4                                       | 43.9  | 8.8                             | 11.4  | 0.19  | 0.26           |
| 235             | 0  | 3.0   | 30.3                                       | 19.2  | 10.6                            | 8.0   | 0.35  | 0.42           |
| 236             | 0  | 1.5   | 42.5                                       | 20.0  | 6.2                             | 9.8   | 0.15  | 0.49           |
| 237             | 0  | 2.5   | 38.0                                       | 18.6  | 11.4                            | 11.4  | 0.30  | 0.67           |
| 238             | 0  | 2.9   | 31.9                                       | 11.9  | 15.0                            | 5.4   | 0.47  | 0.45           |
| 339             | 0  | 4.1   | 59.6                                       | 26.9  | 9.8                             | 5.4   | 0.16  | 0.70           |
| 11              | 0  | 1.5   | 27.4                                       | 33.4  | 6.2                             | 7.0   | 0.22  | 0.21           |
| 12              | 0  | 2.4   | 31.7                                       | 21.6  | 5.5                             | 4.0   | 0.17  | 0.19           |
| 13              | 0  | 2.0   | 36.6                                       | 24.8  | 6.5                             | 6.2   | 0.18  | 0.24           |
| 14              | 0  | 1.2   | 25.3                                       | 22.1  | 3.2                             | 5.7   | 0.13  | 0.26           |
| 15              | 0  | 0.8   | 25.4                                       | 17.7  | 7.0                             | 5.7   | 0.28  | 0.32           |
| 16              | 0  | 1.5   | 24.3                                       | 29.1  | 4.2                             | 4.2   | 0.17  | 0.14           |
| 17              | 0  | 1.4   | 35.3                                       | 22.4  | 7.0                             | 5.7   | 0.70  | 0.25           |
| 18              | 0  | 1.5   | 52.0                                       | 32.5  | 6.7                             | 4.0   | 0.13  | 0.12           |
| mean            | 0  | 2.3   | 36.7                                       | 24.9  | 8.1                             | 8.1   | 0.22  | 0.34           |
| s d             | 0  | 1.1   | 8.7  | 6.8   | 3.4                             | 3.3   | 0.09  | 0.15           |
| error of mean   | 0  | 0.91  | 1.64                                       | 1.29  | 0.65                            | 0.63  | 0.017   | 0.029          |

) Fractured during preparation of specimen



Table 5 Values noted for 30 day old fractures

| animal No     | cross section of marrow in mm <sup>2</sup> |        | to a section of ratio in mm <sup>2</sup> |        | absolute ten de strength in kgf |        | specific tensile strength in kg/mm <sup>2</sup> |                   |
|---------------|--|--------|--|--------|---------------------------------|--------|---|-------------------|
|               | unnailed                                   | nailed | unnailed                                 | nail d | unnailed                        | nailed | unnailed  | nailed            |
| 100           | 0  | 3.6    | 25.3                                     | 25.3   | 15.0                            | 16.0   | 0.59  | 0.63              |
| 101           | 0  | 2.4    | 31.1                                     | 21.2   | 22.0                            | 1.0    | 0.61  | 0.71              |
| 102           | 0  | 4.2    | 2.7                                      | 26.8   | 1.0                             | 16.0   | 0.51  | 0.60              |
| 107           | 0  | 1.7    | 27.2                                     | 20.8   | 20.0                            | 9.0    | 0.71  | 0.43 <sup>1</sup> |
| 108           | 0  | 2.3    | 29.1                                     | 26.1   | 16.0                            | 20.0   | 0.51  | 0.7*              |
| 109           | 2.1  | 2.2    | 26.2                                     | 22.5   | 39.6                            | 2.0    | 1.1   | 0.98 <sup>1</sup> |
| 126           | 1.8  | 2.1    | 21.5                                     | 17.8   | 2.0                             | 30.0   | 1.07  | 1.62              |
| 137           | 1.7  | 1.8    | 28.8                                     | 21.1   | 19.5                            | 11.2   | 0.68  | 0.7               |
| 138           | 0  | 2.2    | 22.9                                     | 20.2   | 1.6                             | 9.8    | 1.07  | 0.37              |
| 139           | 0  | 2.8    | 23.8                                     | 19.1   | 12.1                            | 6.2    | 0.7   | 0.32              |
| 206           | 0  | 3.6    | 31.2                                     | 21.2   | 11                              | 21.2   | 0.77  | 1.00              |
| 207           | 0  | 1.4    | 36.9                                     | 23.1   | 15.0                            | 23.0   | 0.11  | 1.60              |
| 208           | 0  | 3.2    | 30.8                                     | 17.9   | 9.8                             | 11.5   | 0.7   | 0.61              |
| 209           | 0  | 2.6    | 40.5                                     | 23.5   | 8.8                             | 16     | 0.1   | 1.0               |
| 264           | 0  | 2.5    | 2.5                                      | 13.1   | 30.0                            | 20.2   | 1.33  | 1.1               |
| 31            | 0  | 2.1    | 22.8                                     | 2.1    | 13.2                            | 19.7   | 0.58  | 0.77              |
| 39            | 0  | 2.3    | 27.5                                     | 26.0   | 16.0                            | 11.2   | 0.58  | 0.7               |
| 40            | 0  | 2.2    | 30.0                                     | 27.5   | 16.0                            | 11.5   | 0.1   | 0.1 <sup>1</sup>  |
| 41            | 0  | 1.0    | 29.3                                     | 17.7   | 21.6                            | 18.6   | 0.81  | 1.0               |
| 42            | 0  | 0.9    | 2.1                                      | 27.1   | 21.2                            | 21.2   | 0.81  | 0.77              |
| 43            | 0.3  | 1.8    | 27.0                                     | 22.8   | 29.0                            | 19.5   | 1.07  | 0.70              |
| 44            | 0  | 0.7    | 29.6                                     | 21.3   | 7.2                             | 13.5   | 0.21  | 0.92              |
| 45            | 0  | 2.7    | 28.2                                     | 11.3   | 15.0                            | 18.6   | 0.39  | 0.1               |
| 47            | 0  | 3.0    | 27.0                                     | 18.6   | 19.5                            | 11.5   | 0.12  | 0.62              |
| mean          | 0.2  | 2.3    | 28.7                                     | 23.0   | 18.5                            | 17.1   | 0.68  | 0.78              |
| s.d.          | 0.6  | 0.9    | 5.2                                      | 4.7    | 7.4                             | 6      | 0.33  | 0.13              |
| error of mean | 0.13                                       | 0.17   | 1.06                                     | 0.91   | 1.3                             | 1.11   | 0.068   | 0.019             |

\*) Bones with corrosion of nail

Table 6 *t* values noted for 10 day old fractures

| animal No     | cross section of marrow in mm <sup>2</sup> |        | cross section of callus in mm <sup>2</sup> |        | absolute tensile strength in kg |        | specific tensile strength in kg/mm <sup>2</sup> |                  |
|---------------|--|--------|--|--------|---------------------------------|--------|---|------------------|
|               | unnailed                                   | nailed | unnailed                                   | nailed | unnailed                        | nailed | unnailed  | nailed           |
| 84            | 11   | 18     | 131  | 162    | 200                             | 330    | 153   | 204              |
| 85            | 27   | 37     | 203  | 147    | 280                             | 95     | 138   | 065              |
| 87            | 19   | 29     | 252  | 156    | 160                             | 95     | 063   | 061              |
| 88            | 11   | 20     | 181  | 178    | 336                             | 360    | 182   | 202              |
| 89            | 23   | 48     | 208  | 262    | 190                             | 105    | 058   | 040              |
| 91            | 20   | 32     | 335  | 161    | 280                             | 190    | 084   | 118              |
| 92            | 17   | 21     | 256  | 209    | 385                             | 265    | 150   | 127              |
| 93            | 21   | 26     | 236  | 248    | 330                             | 295    | 140   | 119 <sup>4</sup> |
| 94            | 15   | 24     | 208  | 166    | 305                             | 305    | 147   | 184 <sup>4</sup> |
| 266           | 0  | 31     | 224  | 161    | 250                             | 105    | 112   | 065              |
| 267           | 25   | 26     | 229  | 165    | 280                             | 295    | 122   | 179              |
| 268           | 11   | 20     | 205  | 227    | 330                             | 250    | 161   | 110              |
| 269           | 17   | 19     | 173  | 181    | 410                             | 250    | 237   | 138              |
| 270           | 17   | 18     | 217  | 245    | 175                             | 240    | 081   | 098              |
| 271           | 44   | 21     | 219  | 210    | 425                             | 295    | 194   | 140              |
| 272           | 0  | 27     | 241  | 211    | 200                             | 240    | 083   | 114              |
| 21            | 0  | 19     | 271  | 254    | 225                             | 120    | 083   | 047              |
| 24            | 0  | 12     | 284  | 282    | 225                             | 210    | 079   | 074              |
| 25            | 0  | 19     | 215  | 140    | 160                             | 120    | 074   | 086              |
| 26            | 0  | 17     | 266  | 152    | 225                             | 225    | 085   | 148              |
| 27            | 15   | 19     | 224  | 167    | 190                             | 280    | 085   | 168              |
| 28            | 0  | 10     | 230  | 308    | 305                             | 200    | 133   | 065              |
| 29            | 07   | 12     | 295  | 322    | 160                             | 265    | 054   | 082              |
| 30            | 21   | 19     | 365  | 211    | 135                             | 175    | 037   | 083              |
| mean          | 13   | 23     | 236  | 205    | 254                             | 221    | 114   | 113              |
| s d           | 11   | 08     | 50   | 53     | 87                              | 80     | 050   | 049              |
| error of mean | 023  | 017    | 102  | 108    | 177                             | 163    | 0102  | 0099             |

<sup>4</sup>) Bones with corrosion of nail

Table 7 Values noted for 50 day old fractures

| animal No | cross section of marrow in mm <sup>2</sup> |       | cross section of callus in mm <sup>2</sup> |       | at suture tensile strength in kg |       | specific tensile strength in kg/mm <sup>2</sup> |       |
|-----------|--|-------|--|-------|----------------------------------|-------|---|-------|
|           | unaided                                    | noded | unaided                                    | noded | unaided                          | noded | unaided   | noded |
| 95        | 31   | 34    | 26.2                                       | 30.0  | 12.5                             | 0.18  | 1.23  | 0.18  |
| 96        | 29   | 20    | 16.7                                       | 18.0  | 23.0                             | 1.38  | 1.01  | 1.38  |
| 97        | 36   | 25    | 14.9                                       | 17.2  | 21.5                             | 1.25  | 3.23  | 1.25  |
| 98        | 30   | 22    | 19.4                                       | 19.9  | 26.5                             | 1.37  | 2.37  | 1.37  |
| 124       | 1  | 37    | 18.9                                       | 18.1  | 33                               | 1.77  | 1.77  | 1.71  |
| 125       | 38   | 25    | 18.9                                       | 17.6  | 33.5                             | 1.90  | 2.62  | 1.90  |
| 126       | 18   | 26    | 17.6                                       | 20.0  | 39.0                             | 1.95  | 81  | 1.95  |
| 127       | 10   | 18    | 16.1                                       | 16.1  | 18                               | 1.43  | 1.10  | 1.43  |
| 184       | 0.0  | 27    | 20.1                                       | 21.5  | 31.0                             | 1.90  | 0.86  | 1.90  |
| 185       | 21   | 19    | 17.9                                       | 11    | 26.5                             | 1.11  | 1.11  | 1.11  |
| 186       | 10   | 21    | 16.7                                       | 8.5   | 17                               | 1.08  | 1.1   | 1.08  |
| 187       | 0  | 29    | 17.1                                       | 16.5  | 20                               | 1.57  | 0.9   | 1.57  |
| 188       | 21   | 14    | 17.3                                       | 21.0  | 30.0                             | 1.81  | 1.31  | 1.81  |
| 189       | 20   | 27    | 23.8                                       | 18.1  | 30.0                             | 1.73  | 1.37  | 1.73  |
| 190       | 31   | 31    | 17.8                                       | 14.0  | 30.0                             | 1.90  | 2.47  | 1.90  |
| 191       | 31   | 23    | 23.5                                       | 19.3  | 11                               | 1.71  | 0.31  | 1.71  |
| 76        | 25   | 19    | 19.2                                       | 20.7  | 41.0                             | 1.1   | 1   | 1.1   |
| 77        | 22   | 28    | 13.6                                       | 13.3  | 28                               | 2.24  | 1.4   | 2.24  |
| 78        | 17   | 11    | 13.7                                       | 14.6  | 30.0                             | 1.19  | 1.31  | 1.19  |
| 79        | 18   | 07    | 6.0  | 15.6  | 11.0                             | 0.87  | 1.8   | 0.87  |
| 80        | 13   | 12    | 17.0                                       | 14.1  | 37                               | 2.37  | 2.1   | 2.37  |
| 81        | 09   | 13    | 23.7                                       | 15.2  | 30.0                             | 1.37  | 1.27  | 1.37  |
| 82        | 19   | 12    | 16.0                                       | 18.3  | 21.0                             | 0.91  | 1.50  | 0.91  |
| 83        | 26   | 17    | 20.4                                       | 20.7  | 11.0                             | 0.91  | 2.01  | 0.91  |
| mean      | 22   | 21    | 17.9                                       | 31.1  | 26.7                             | 1.2   | 1.77  | 1.2   |
| s.d.      | 11   | 0.8   | 2.8  | 11.2  | 7.7                              | 0.47  | 0.70  | 0.47  |
| error     |  |       |  |       |                                  |       |   |       |
| of mean   | 0.22                                       | 0.16  | 0.58                                       | 1.17  |                                  |       | 0.142   | 0.096 |

a) Bones with corrosion of nail

Table 8 Values not 1 for 60 d<sub>13</sub> old fractures

| animal<br>No     | cross section of marrow<br>in mm <sup>2</sup> |        | cross section of collar<br>in mm <sup>2</sup> |        | ab olute tensile strength<br>in kg <sub>s</sub> |        | specific tensile strength<br>in kg/mm <sup>2</sup> |                  |
|------------------|---|--------|---|--------|---|--------|--|------------------|
|                  | unnailed                                      | nailed | unnailed                                      | nailed | unnailed  | nailed | unnailed   | nailed           |
| 116              | 18  | 10     | 231   | 116    | 415   | 375    | 193  | 257 <sup>a</sup> |
| 117              | 38  | 11     | 137   | 206    | 515   | 285    | 376  | 138              |
| 118              | 12  | 09     | 100   | 160    | 550   | 300    | 341  | 188              |
| 119              | 78  | 02     | 203   | 151    | 325   | 285    | 175  | 189              |
| 120              | 21  | 31     | 218   | 212    | 250   | 195    | 100  | 092 <sup>a</sup> |
| 130              | 12  | 20     | 228   | 233    | 365   | 565    | 218  | 212 <sup>a</sup> |
| 131              | 35  | 30     | 231   | 223    | 520   | 375    | 238  | 168              |
| 216              | 12  | 23     | 133   | 145    | 300   | 300    | 226  | 207              |
| 247              | 28  | 12     | 125   | 118    | 390   | 250    | 312  | 212              |
| 248              | 16  | 15     | 95  | 100    | 175   | 70     | 181  | 070              |
| 251              | 72  | 35     | 121   | 167    | 265   | 145    | 219  | 087              |
| 252              | 11  | 36     | 131   | 124    | 600   | 375    | 158  | 302              |
| 253              | 38  | 31     | 151   | 165    | 355   | 180    | 231  | 290              |
| 254              | 22  | 12     | 139   | 114    | 195   | 160    | 140  | 111              |
| 255              | 18  | 37     | 110   | 131    | 285   | 285    | 259  | 218              |
| 280              | 30  | 36     | 298   | 171    | 390   | 195    | 131  | 111              |
| 281              | 41  | 10     | 151   | 175    | 460   | 300    | 299  | 171              |
| 282              | 51  | 17     | 115   | 145    | 125   | 230    | 150  | 159              |
| 67               | 29  | 11     | 146   | 191    | 300   | 300    | 205  | 157              |
| 68               | 36  | 13     | 182   | 200    | 600   | 415    | 330  | 223              |
| 70               | 29  | 11     | 170   | 205    | 285   | 285    | 168  | 139              |
| 71               | 51  | 34     | 142   | 144    | 280   | 150    | 197  | 104              |
| 74               | 11  | 11     | 111   | 124    | 320   | 320    | 227  | 258              |
| 75               | 90  | 12     | 116   | 162    | 320   | 300    | 220  | 185 <sup>a</sup> |
| mean             | 32  | 23     | 161   | 161    | 382   | 290    | 211  | 178              |
| s d              | 15  | 12     | 51  | 35     | 128   | 112    | 986  | 065              |
| error<br>of mean | 0.30  | 0.24   | 1.03  | 0.72   | 2.61  | 2.28   | 0.176  | 0.132            |

<sup>a</sup>) Bones with corrosion of nail

Table 9 *Values noted for 70-day old fractures*

| animal<br>No.    | cross section of marrow<br>in mm <sup>2</sup> |           | cross section of callus<br>in mm <sup>2</sup> |           | absolute tensile strength<br>in kg |           | specific tensile strength<br>in kg/mm <sup>2</sup> |           |
|------------------|---|-----------|---|-----------|------------------------------------|-----------|--|-----------|
|                  | unfractured                                   | fractured | unfractured                                   | fractured | unfractured                        | fractured | unfractured  | fractured |
| 132              | 20  | 13        | 19.5  | 17.1      | 56.5                               | 41.0      | 2.11   | 3.32      |
| 131              | 33  | 18        | 22.8  | 21.7      | 49.5                               | 30.0      | 1.33   | 2.29      |
| 134              | 32  | 29        | 29.1  | 17.8      | 32.0                               | 53.0      | 2.11   | 1.85      |
| 135              | 33  | 27        | 18.8  | 20.5      | 60.0                               | 49.5      | 0.67   | 2.91      |
| 140              | 69  | 40        | 19.1  | 1.8       | 30.0                               | 13.5      | 0.36   | 1.90      |
| 141              | 39  | 38        | 18.1  | 1.1       | 11                                 | 11.5      | 0.41   | 0.33      |
| 142              | 1   | 19        | 0.3   | 17.1      | 1.5                                | 41        | 0.19   | 3.00      |
| 143              | 53  | 36        | 2.1   | 1.0       | 21                                 | 5.0       | 0.41   | 1.50      |
| 157              | 59  | 56        | 0.1   | 1.5       | 1                                  | 1         | 1.66   | 0.12      |
| 257              | 51  | 0.1       | 1   | 11.8      | 19.5                               | 13.0      | 2.11   | 1.15      |
| 258              | 34  | 15        | 11.1  | 11.1      | 0.5                                | 0.5       | 2.39   | 0.07      |
| 259              | 31  | 35        | 9.7   | 11.1      | 3.0                                | 4         | 4.38   | 2.15      |
| 260              | 56  | 1         | 18.1  | 11.3      | 11.5                               | 11.5      | 16   | 3.11      |
| 262              | 45  | 45        | 14.0  | 14.1      | 0.5                                | 0.5       | 1.77   | 0.13      |
| 263              | 39  | 28        | 13.3  | 1.9       | 3.0                                | 1.5       | 3.06   | 0.1       |
| 264              | 19  | 0.8       | 13.1  | 12.1      | 3.0                                | 3.0       | 0.76   | 3.15      |
| 283              | 39  | 18        | 13.0  | 17.1      | 16.0                               | 16.0      | 3.1  | 1.8       |
| 38               | 13  | 1         | 11.1  | 20.8      | 3.0                                | 3.0       | 0.1  | 1.1       |
| 59               | 92  | 34        | 19.0  | 1.8       | 7.0                                | 7.0       | 1.83   | 1         |
| 60               | 44  | 95        | 25.1  | 13.9      | 1.0                                | 31.0      | 1.33   | 1.5       |
| 61               | 62  | 18        | 10.5  | 16.6      | 11.0                               | 39.0      | 2.07   | 2.07      |
| 65               | 13  | 15        | 11.6  | 15.6      | 30.0                               | 31.0      | 3.1  | 1.93      |
| 65               | 25  | 37        | 13.0  | 11.1      | 0.0                                | 10.0      | 2.33   | 4.8       |
| 66               | 16  | 15        | 17.6  | 0.1       | 18.0                               | 15.0      | 7  | 0.36      |
| mean             | 16  | 31        | 17.2  | 1.8       | 36.5                               | 33.3      | 2.37   | 0.33      |
| s.d.             | 16  | 19        | 1.5   | 3.1       | 12.0                               | 8.1       | 0.6  | 0.10      |
| error<br>of mean | 0.33  | 0.38      | 0.85  | 0.62      | 1.61                               | 1.61      | 0.13   | 0.15      |

1) Fractured during preparation of specimen

2) Bones with cortical defect

Table 10 Values noted for 80 day old fractures

| animal No     | cross section of marrow in mm <sup>2</sup> |       | cross section of callus in mm <sup>2</sup> |       | absolute tensile strength in kg |       | specific tensile strength in kg/mm <sup>2</sup> |                   |
|---------------|--|-------|--|-------|---------------------------------|-------|---|-------------------|
|               | unnailed                                   | naild | unnailed                                   | naild | unnailed                        | naild | unnailed  | naild             |
| 144           | 4.5  | 2.3   | 21.4                                       | 16.6  | 53.0                            | 41.0  | 2.48  | 2.47              |
| 145           | 5.2  | 1.7   | 18.6                                       | 26.8  | 30.0                            | 39.0  | 1.61  | 1.46              |
| 146           | 3.6  | 1.6   | 18.7                                       | 16.1  | 46.0                            | 48.0  | 2.46  | 2.98              |
| 147           | 2.9  | 2.6   | 15.8                                       | 14.6  | 41.0                            | 33.5  | 2.59  | 2.29              |
| 192           | 3.8  | 4.2   | 17.2                                       | 15.2  | 54.0                            | —     | 3.14  | — <sup>1</sup>    |
| 193           | 4.4  | 4.0   | 18.6                                       | 18.9  | 41.0                            | —     | 2.20  | — <sup>1</sup>    |
| 194           | 5.0  | 3.5   | 21.4                                       | 17.2  | 41.0                            | 37.0  | 1.92  | 2.15              |
| 196           | 2.9  | 2.6   | 21.0                                       | 21.1  | 58.0                            | 60.0  | 2.76  | 2.84              |
| 197           | 5.1  | 2.3   | 21.8                                       | 14.9  | 37.0                            | 35.0  | 1.70 <sup>2</sup>                               | 2.35              |
| 198           | 4.6  | 3.0   | 25.0                                       | 20.5  | 52.0                            | 48.0  | 2.08  | 2.34              |
| 199           | 5.0  | 4.1   | 14.9                                       | 19.0  | 20.0                            | 52.0  | 1.34  | 2.74              |
| 200           | 4.2  | 5.6   | 13.8                                       | 21.5  | 41.0                            | 41.0  | 2.97  | 1.91              |
| 202           | 4.6  | 1.5   | 17.7                                       | 17.4  | 37.0                            | 52.0  | 2.69  | 3.00              |
| 203           | 2.9  | 2.8   | 18.2                                       | 18.4  | 58.0                            | 67.0  | 3.19  | 3.64              |
| 273           | 2.8  | 3.6   | 14.9                                       | 14.9  | 48.0                            | 28.5  | 3.22  | 1.91              |
| 274           | 4.7  | 3.8   | 16.0                                       | 14.6  | 53.0                            | 32.0  | 3.32 <sup>2</sup>                               | 2.19 <sup>2</sup> |
| 275           | 2.9  | 3.3   | 14.9                                       | 12.3  | 51.5                            | 37.5  | 3.46  | 3.05              |
| 48            | 4.4  | 3.7   | 15.3                                       | 14.8  | 50.0                            | 32.0  | 3.27  | 2.16              |
| 50            | 5.2  | 7.5   | 14.4                                       | 13.8  | 41.0                            | 35.0  | 2.84  | 2.54              |
| 51            | 2.0  | 1.1   | 14.4                                       | 13.8  | 35.0                            | 32.0  | 2.43  | 2.32              |
| 53            | 3.1  | 2.5   | 15.8                                       | 20.0  | 34.0                            | 39.0  | 2.15 <sup>2</sup>                               | 1.95              |
| 54            | 4.7  | 1.4   | 12.9                                       | 15.5  | 26.0                            | 30.0  | 2.02  | 1.94              |
| 56            | 3.2  | 1.7   | 14.0                                       | 12.8  | 39.0                            | 26.0  | 2.79 <sup>2</sup>                               | 2.02              |
| 57            | 3.2  | 2.0   | 14.6                                       | 15.0  | 32.0                            | 26.0  | 2.19  | 1.73              |
| mean          | 4.0  | 3.0   | 17.1                                       | 16.9  | 42.4                            | 39.6  | 2.51  | 2.36              |
| s d           | 1.0  | 1.5   | 3.1  | 3.4   | 10.1                            | 10.9  | 0.59  | 0.51              |
| error of mean | 0.19                                       | 0.30  | 0.64                                       | 0.69  | 2.07                            | 2.31  | 0.121   | 0.109             |

<sup>1</sup>) Fractured during preparation of specimen<sup>2</sup>) Bones with cortical defect

Table 11 Values noted for 100 day old fractures

| animal no | cross section of marrow in mm <sup>2</sup> |         | cross section of callus in mm <sup>2</sup> |           | absolute tensile strength in kg |        | specific tensile strength in kg/mm <sup>2</sup> |                   |
|-----------|--|---------|--|-----------|---------------------------------|--------|---|-------------------|
|           | unsealed                                   | sealed  | unsealed                                   | sealed    | unsealed                        | sealed | unsealed  | sealed            |
| 168       | 29   | 71      | 177  | 168       | 67.0                            | 73.0   | 3.79 <sup>2</sup>                               | 4.34 <sup>1</sup> |
| 170       | 66   | 30      | 203  | 212       | 51.0                            | 28.0   | 2.66 <sup>2</sup>                               | 1.32 <sup>2</sup> |
| 171       | 37   | 25      | 136  | 116       | 57.0                            | 41.0   | 1.26 <sup>2</sup>                               | 2.81 <sup>2</sup> |
| 172       | 25   | 21      | 162  | 110       | 39.0                            | 39.0   | 2.41 <sup>2</sup>                               | 3.52              |
| 173       | 28   | 52      | 176  | 146       | 43.0                            | 32.0   | 2.41 <sup>2</sup>                               | 2.19              |
| 174       | 46   | 20      | 136  | 109       | 22.0                            | 30.0   | 1.62 <sup>2</sup>                               | 3.21 <sup>2</sup> |
| 175       | 33   | 53      | 217  | 197       | 15.0                            | 58.0   | 2.07 <sup>2</sup>                               | 2.91 <sup>2</sup> |
| 176       | 45   | 34      | 196  | 160       | 41.0                            | 56.0   | 2.09 <sup>2</sup>                               | 3.03 <sup>2</sup> |
| 177       | 37   | 23      | 201  | 224       | 10                              | 28.0   | 2.11 <sup>2</sup>                               | 1.25 <sup>2</sup> |
| 178       | 44   | 30 (37) | 178  | 168 (115) | 43.0                            | 48.0   | 2.10 <sup>2</sup>                               | 1.21 <sup>2</sup> |
| 179       | 67   | 45      | 187  | 178       | 38.0                            | 71.0   | 2.5   | 1.00              |
| 180       | 28   | 16 (29) | 172  | 212 (107) | 15.0                            | 10.0   | 2.01 <sup>2</sup>                               | 1.1               |
| 181       | 45   | 34      | 105  | 113       | 40                              | 30     | 1.1   | 2.71              |
| 182       | 55   | 51      | 210  | 205       | 0                               | 50.0   | 3.1   | 5.00 <sup>2</sup> |
| 183       | 48   | 29 (60) | 236  | 183 (106) | 43.0                            | 11.0   | 1.1   | 1.10 <sup>2</sup> |
| 222       | 40   | 16      | 111  | 106       | 41.0                            | 17.0   | 1.1   | 1.1               |
| 224       | 16   | 34      | 160  | 146       | 0                               | 4.0    | 2.1   | 1.1               |
| 225       | 44   | 13      | 130  | 132       | 32.0                            | 4.0    | 1.1   | 1.1               |
| 226       | 56   | 38      | 108  | 100       | 50.0                            | 41.0   | 1.1   | 1.1               |
| 227       | 60   | 27      | 190  | 127       | 57.0                            | 53.0   | 1.1   | 1.1               |
| 228       | 61   | 60      | 131  | 157       | 33                              | 30.0   | 1.1   | 1.1               |
| 229       | 27   | 37      | 101  | 102       | 30.0                            | 0.0    | 1.1   | 1.1               |
| 276       | 31   | 40      | 123  | 138       | 67.0                            | 41.0   | 4   | 2.1               |
| 277       | 00   | 38      | 150  | 161       | 63.0                            | 3.0    | 4.20 <sup>2</sup>                               | 1.7 <sup>2</sup>  |
| mean      | 44   | 39      | 163  | 149       | 47.8                            | 4.0    | 3.10  | 1.02              |
| s.d.      | 13   | 13      | 40   | 38        | 119                             | 13.1   | 1.31  | 1.0               |
| cor       |  |         |  |           | 3.0                             | 2.89   | 0.270   | 0.30              |
| mean      | 0.26                                       | 0.23    | 0.81                                       | 0.81      |                                 |        |   |                   |

ues with cortical defect

ues fractured outside callus area Figures in brackets denote cross section of bone at site of fracture

ues bones are not included in the analysis

ues w.d. corrosion of nail

Table 12. Values noted for 190 day old fractures

| animal No       | cross section of marrow in mm <sup>2</sup> |        | cross section of callus in mm <sup>2</sup> |          | absolute tensile strength in kg |        | specific tensile strength in kg/mm <sup>2</sup> |                  |
|-----------------|--|--------|--|----------|---------------------------------|--------|---|------------------|
|                 | unnailed                                   | nailed | unnailed                                   | nailed   | unnailed                        | nailed | unnailed  | nailed           |
| 148             | 49   | 76     | 191  | 153      | 220                             | 390    | 115 <sup>a</sup>                                | 255 <sup>a</sup> |
| 149             | 43   | 35     | 223  | 213      | 580                             | 390    | 260   | 183 <sup>a</sup> |
| 150             | 66   | 42     | 160  | 175      | 600                             | 410    | 375 <sup>a</sup>                                | 234              |
| 151             | 38   | 19     | 197  | 148      | 690                             | 540    | 350   | 365              |
| 156             | 65   | 29(57) | 186  | 188(123) | 410                             | 500    | 220   | 407 <sup>a</sup> |
| 157             | 41   | 33     | 162  | 183      | 580                             | 630    | 358   | 344              |
| 158             | 46   | 43     | 138  | 10       | 410                             | 650    | 297 <sup>a</sup>                                | 382 <sup>a</sup> |
| 159             | 82   | 77     | 210  | 241      | —                               | 650    | —   | 270 <sup>a</sup> |
| 165             | 38   | 27     | 171  | 143      | 650                             | 750    | 380 <sup>a</sup>                                | 525              |
| 166             | 26   | 15     | 186  | 162      | 370                             | 220    | 200 <sup>a</sup>                                | 136 <sup>a</sup> |
| 167             | 37   | 23     | 134  | 167      | 720                             | 630    | 537 <sup>a</sup>                                | 377 <sup>a</sup> |
| 210             | 67   | 57     | 124  | 146      | 520                             | 560    | 419 <sup>a</sup>                                | 342 <sup>a</sup> |
| 211             | 57   | 58     | 124  | 149      | 450                             | 320    | 362   | 214 <sup>a</sup> |
| 21 <sup>a</sup> | 48   | 38     | 119  | 114      | 410                             | 300    | 345 <sup>a</sup>                                | 263 <sup>a</sup> |
| 213             | 40   | 26     | 144  | 137      | 480                             | 580    | 333 <sup>a</sup>                                | 423 <sup>a</sup> |
| 214             | 43   | 26     | 124  | 154      | 450                             | 390    | 363 <sup>a</sup>                                | 253 <sup>a</sup> |
| 215             | 53   | 25     | 130  | 137      | 480                             | 540    | 369 <sup>a</sup>                                | 394 <sup>a</sup> |
| 216             | 62   | 48     | 139  | 134      | 520                             | 410    | 374 <sup>a</sup>                                | 306 <sup>a</sup> |
| 218             | 45   | 25     | 156  | 134      | 320                             | 370    | 205   | 276              |
| 219             | 43   | 24     | 93   | 101      | 450                             | 430    | 484 <sup>a</sup>                                | 426              |
| 220             | 32   | 31     | 108  | 117      | 280                             | 260    | 259 <sup>a</sup>                                | 222              |
| 221             | 27   | 17     | 129  | 110      | 370                             | 320    | 287 <sup>a</sup>                                | 290 <sup>a</sup> |
| 278             | 65   | 26     | 121  | 129      | 520                             | 480    | 430   | 372 <sup>a</sup> |
| 279             | 52   | 35     | 167  | 127      | 390                             | 450    | 234 <sup>a</sup>                                | 354 <sup>a</sup> |
| mean            | 49   | 36     | 152  | 150      | 473                             | 464    | 329   | 313              |
| s.d.            | 14   | 17     | 34   | 34       | 126                             | 142    | 997   | 991              |
| error of mean   | 0.28                                       | 0.35   | 0.69                                       | 0.67     | 2.63                            | 2.95   | 0.903   | 0.189            |

1) Fractured during preparation of specimen

2) Bones with cortical defect

3) Bones fractured outside callus area. Figures in brackets denote cross section of bone at site of fracture

These bones are not included in the analysis

4) Bones with corrosion of nail



Table 13 Values noted for 10 day old fractures

| animal<br>No     | wt of bone ash in mg |        |          |        |          |        | 5 <sup>90</sup> activity in % of dose $\times 10^2$ |        |          |        |          |        |
|------------------|----------------------|--------|----------|--------|----------|--------|---|--------|----------|--------|----------|--------|
|                  | A part               |        | B part   |        | C part   |        | A part  |        | B part   |        | C part   |        |
|                  | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed  | nailed | unnailed | nailed | unnailed | nailed |
| 103              | 134                  | 168    | 107      | 99     | 198      | 231    | 164   | 216    | 99       | 93     | 72       | 104    |
| 104              | 144                  | 178    | 80       | 61     | 199      | 227    | 144   | 228    | 72       | 16     | 71       | 112    |
| 10               | 141                  | 169    | 96       | 116    | 268      | 288    | 120   | 185    | 52       | 77     | 61       | 7      |
| 106              | 166                  | 188    | 100      | 89     | 260      | 288    | 150   | 212    | 51       | 46     | 80       | 112    |
| 110              | 141                  | 165    | 87       | 106    | 243      | 215    | 126   | 190    | 66       | 77     | 91       | 90     |
| 111              | 154                  | 180    | 103      | 113    | 227      | 244    | 119   | 258    | 91       | 111    | 89       | 83     |
| 112              | 119                  | 147    | 108      | 94     | 374      | 365    | 80  | 139    | 58       | 51     | 50       | 59     |
| 113              | 171                  | 203    | 111      | 107    | 301      | 346    | 116   | 176    | 64       | 62     | 53       | 93     |
| 160              | 167                  | 139    | 133      | 122    | 253      | 266    | 61  | 106    | 39       | 43     | 37       | 33     |
| 161              | 10                   | 120    | 139      | 146    | 254      | 270    | 80  | 127    | 65       | 81     | 56       | 57     |
| 162              | 103                  | 106    | 129      | 121    | 230      | 253    | 33  | 67     | 42       | 45     | 35       | 39     |
| 163              | 151                  | 198    | 190      | 220    | 300      | 299    | 84  | 81     | 88       | 97     | 50       | 68     |
| 240              | 138                  | 143    | 130      | 131    | 290      | 291    | 88  | 90     | 67       | 100    | 80       | 82     |
| 241              | 117                  | 139    | 135      | 117    | 300      | 330    | 15  | 109    | 10       | 6      | 28       | 41     |
| 242              | 118                  | 164    | 121      | 121    | 212      | 209    | 29  | 115    | 61       | 68     | 42       | 58     |
| 243              | 137                  | 155    | 118      | 112    | 191      | 200    | 118   | 191    | 87       | 115    | 57       | 74     |
| 244              | 199                  | 141    | 129      | 130    | 270      | 318    | 141   | 173    | 95       | 91     | 70       | 106    |
| mean             | 138                  | 160    | 121      | 122    | 258      | 275    | 165   | 167    | 67       | 75     | 60       | 76     |
| s.d.             | 19                   | 26     | 96       | 31     | 49       | 46     | 39  | 56     | 19       | 26     | 19       | 20     |
| error<br>of mean | 17                   | 63     | 64       | 82     | 118      | 112    | 091   | 137    | 047      | 063    | 046      | 062    |

Table 14 Values noted for 20 day old fractures

| animal<br>No | wt of bone ash in mg |        |          |        |          |        | Sr <sup>85</sup> activity in % of dose $\times 10^4$ |        |          |        |          |        |
|--------------|----------------------|--------|----------|--------|----------|--------|--|--------|----------|--------|----------|--------|
|              | A part               |        | B part   |        | C part   |        | A part   |        | B part   |        | C part   |        |
|              | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed   | nailed | unnailed | nailed | unnailed | nailed |
| 114          | 151                  | 179    | 159      | 153    | 267      | 262    | 86   | 120    | 104      | 90     | 45       | 55     |
| 120          | 161                  | 178    | 188      | 167    | 199      | 248    | 120  | 210    | 224      | 192    | 54       | 130    |
| 151          | 135                  | 191    | 215      | 186    | 202      | 212    | 176  | 206    | 284      | 200    | 72       | 76     |
| 155          | 150                  | 165    | 230      | 187    | 226      | 253    | 132  | 145    | 227      | 147    | 54       | 91     |
| 201          | 140                  | 182    | 145      | 237    | 275      | 292    | 122  | 216    | 164      | 107    | 85       | 120    |
| 205          | 156                  | 193    | 194      | 160    | 281      | 262    | 240  | 366    | 408      | 318    | 117      | 135    |
| 230          | 173                  | 191    | 231      | 191    | 260      | 296    | 180  | 212    | 365      | 273    | 98       | 106    |
| 231          | 162                  | 195    | 214      | 169    | 221      | 302    | 101  | 135    | 157      | 100    | 38       | 55     |
| 232          | 151                  | 156    | 179      | 166    | 236      | 243    | 103  | 191    | 226      | 186    | 58       | 73     |
| 233          | 176                  | 197    | 197      | 166    | 234      | 262    | 95   | 156    | 185      | 134    | 57       | 65     |
| 234          | 123                  | 158    | 202      | 115    | 295      | 314    | 57   | 203    | 278      | 275    | 50       | 86     |
|              | 131                  | 151    | 157      | 148    | 223      | 235    | 32   | 38     | 42       | 27     | 16       | 18     |
|              | 119                  | 143    | 217      | 152    | 227      | 268    | 92   | 139    | 204      | 103    | 51       | 57     |
|              | 155                  | 179    | 178      | 147    | 250      | 280    | 34   | 52     | 59       | 28     | 17       | 26     |
|              | 144                  | 225    | 154      | 113    | 192      | 230    | 128  | 124    | 141      | 68     | 47       | 55     |
|              | 150                  | 150    | 92       | 148    | 250      | 262    | 129  | 160    | 194      | 141    | 54       | 120    |
| can          | 151                  | 171    | 184      | 160    | 240      | 264    | 115  | 171    | 204      | 149    | 57       | 79     |
| sd           | 14                   | 22     | 37       | 20     | 30       | 28     | 54   | 79     | 99       | 86     | 26       | 36     |
| err          | 36                   | 56     | 92       | 50     | 76       | 69     | 134  | 197    | 247      | 216    | 066      | 089    |

Table 1 Values not d for 30 d ry old fractures

| animal<br>No     | wet of bone ash in mg |        |          |        |          |        | Sr <sup>87</sup> activity in |        |          |        |          |        | 10 <sup>4</sup>  |  |
|------------------|-----------------------|--------|----------|--------|----------|--------|------------------------------|--------|----------|--------|----------|--------|------------------|--|
|                  | A part                |        | B part   |        | C part   |        | A part                       |        | B part   |        | C part   |        |                  |  |
|                  | unmelted              | melted | unmelted | melted | unmelted | melted | unmelted                     | melted | unmelted | melted | unmelted | melted |                  |  |
| 100              | 132                   | 190    | 164      | 169    | 276      | 357    | 46                           | 116    | 87       | 31     | 87       | 31     | 79               |  |
| 101              | 187                   | 107    | 94       | 183    | 238      | 86     | 166                          | 908    | 56       | 65     | 185      | 65     | 103              |  |
| 102              | 167                   | 117    | 177      | 94     | 1        | 92     | 106                          | 38     | 193      | 73     | 193      | 73     | 90               |  |
| 107              | 191                   | 181    | 183      | 189    | 15       | 910    | 934                          | 14     | 189      | 71     | 189      | 71     | 91 <sup>4</sup>  |  |
| 108              | 209                   | 118    | 190      | 217    | 177      | 966    | 204                          | 214    | 153      | 5      | 153      | 5      | 76 <sup>4</sup>  |  |
| 109              | 184                   | 223    | 180      | 165    | 98       | 90     | 287                          | 317    | 284      | 119    | 284      | 119    | 182 <sup>4</sup> |  |
| 136              | 105                   | 139    | 198      | 156    | 97       | 219    | 138                          | 197    | 173      | 83     | 173      | 83     | 90               |  |
| 137              | 116                   | 163    | 231      | 175    | 177      | 200    | 93                           | 97     | 102      | 50     | 102      | 50     | 71               |  |
| 138              | 145                   | 165    | 193      | 205    | 230      | 250    | 86                           | 88     | 104      | 44     | 104      | 44     | 109              |  |
| 139              | 156                   | 188    | 189      | 164    | 241      | 214    | 90                           | 110    | 100      | 41     | 100      | 41     | 71               |  |
| 166              | 191                   | 215    | 105      | 158    | 267      | 293    | 121                          | 186    | 116      | 40     | 116      | 40     | 57               |  |
| 207              | 194                   | 189    | 229      | 171    | 273      | 290    | 11                           | 137    | 160      | 52     | 160      | 52     | 56               |  |
| 208              | 141                   | 148    | 165      | 130    | 225      | 252    | 195                          | 318    | 266      | 85     | 266      | 85     | 116              |  |
| 209              | 155                   | 170    | 225      | 183    | 36       | 396    | 103                          | 101    | 118      | 10     | 118      | 10     | 51               |  |
| 265              | 174                   | 165    | 166      | 123    | 298      | 280    | 59                           | 50     | 63       | 05     | 63       | 05     | 09               |  |
| mean             | 168                   | 179    | 197      | 175    | 211      | 269    | 113                          | 185    | 160      | 58     | 160      | 58     | 84               |  |
| s.d.             | 28                    | 26     | 35       | 30     | 49       | 50     | 68                           | 97     | 68       | 2      | 68       | 2      | 36               |  |
| error<br>of mean | 73                    | 67     | 89       | 79     | 107      | 133    | 176                          | 250    | 176      | 053    | 176      | 053    | 093              |  |

\*) Bones with corrosion of nail

Table 16 Values noted for 40 day old fractures

| animal No     | wt of bone ash in mg |        |          |        |          |        | St <sup>45</sup> activity in % of dose $\times 10^3$ |        |          |        |          |                  |
|---------------|----------------------|--------|----------|--------|----------|--------|--|--------|----------|--------|----------|------------------|
|               | A part               |        | B part   |        | C part   |        | A part   |        | B part   |        | C part   |                  |
|               | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed   | nailed | unnailed | nailed | unnailed | nailed           |
| 84            | 133                  | 130    | 108      | 95     | 291      | 381    | 94   | 101    | 60       | 59     | 43       | 109              |
| 85            | 174                  | 129    | 156      | 111    | 290      | 380    | 208  | 210    | 152      | 107    | 79       | 214              |
| 87            | 163                  | 189    | 141      | 126    | 352      | 338    | 94   | 113    | 98       | 62     | 51       | 73               |
| 88            | 116                  | 158    | 139      | 120    | 326      | 335    | 58   | 66     | 70       | 59     | 43       | 73               |
| 89            | 189                  | 183    | 176      | 197    | 242      | 284    | 116  | 131    | 62       | 87     | 46       | 54               |
| 91            | 135                  | 171    | 178      | 118    | 225      | 266    | 131  | 170    | 145      | 76     | 43       | 61               |
| 92            | 160                  | 206    | 252      | 178    | 203      | 283    | 119  | 220    | 146      | 112    | 40       | 82               |
| 93            | 159                  | 177    | 195      | 213    | 240      | 251    | 140  | 137    | 150      | 176    | 75       | 94 <sup>4</sup>  |
| 94            | 143                  | 141    | 166      | 161    | 261      | 372    | 76   | 84     | 75       | 62     | 31       | 114 <sup>4</sup> |
| 266           | 149                  | 137    | 157      | 137    | 295      | 317    | 122  | 97     | 151      | 118    | 48       | 91               |
| 267           | 169                  | 159    | 162      | 144    | 237      | 250    | 177  | 177    | 92       | 86     | 70       | 75               |
| 268           | 172                  | 199    | 151      | 149    | 215      | 246    | 249  | 283    | 115      | 128    | 103      | 99               |
| 269           | 170                  | 169    | 149      | 143    | 210      | 231    | 210  | 236    | 109      | 122    | 80       | 104              |
| 270           | 165                  | 158    | 157      | 164    | 257      | 268    | 196  | 199    | 107      | 123    | 99       | 116              |
| 271           | 186                  | 161    | 159      | 139    | 234      | 255    | 238  | 236    | 122      | 143    | 73       | 113              |
| 272           | 149                  | 143    | 184      | 170    | 319      | 347    | 130  | 105    | 133      | 125    | 50       | 63               |
| mean          | 158                  | 163    | 161      | 148    | 262      | 300    | 146  | 160    | 112      | 103    | 61       | 96               |
| s d           | 20                   | 24     | 31       | 32     | 45       | 52     | 56   | 65     | 33       | 34     | 22       | 37               |
| error of mean | 50                   | 59     | 77       | 79     | 112      | 130    | 141  | 162    | 082      | 086    | 055      | 093              |

<sup>4</sup>) Bones with corrosion of nail

Table 1. Values noted for 50 day old fractures

| animal No       | wt of bone in mg |        |                 |        |          |                 | S <sup>35</sup> activity in % of dose $\times 10^3$ |        |          |        |          |                   |
|-----------------|------------------|--------|-----------------|--------|----------|-----------------|---|--------|----------|--------|----------|-------------------|
|                 | A part           |        | B part          |        | C part   |                 | A part  |        | B part   |        | C part   |                   |
|                 | unnailed         | nailed | unnailed        | nailed | unnailed | nailed          | unnailed  | nailed | unnailed | nailed | unnailed | nailed            |
| 9 <sub>a</sub>  | 178              | 183    | 62              | 251    | 277      | 35 <sup>a</sup> | 18.9  | 241    | 9.2      | 30.0   | 11.4     | 31.0 <sup>a</sup> |
| 9 <sub>b</sub>  | 151              | 167    | 17 <sup>a</sup> | 187    | 298      | 279             | 9.9   | 67     | 7.1      | 6.6    | 4.5      | 6.3               |
| 9 <sub>c</sub>  | 13               | 132    | 173             | 202    | 460      | 276             | 9.1   | 111    | 8        | 12.4   | 6.4      | 8.4               |
| 9 <sub>d</sub>  | 132              | 115    | 199             | 190    | 223      | 256             | 16.8  | 238    | 15.4     | 20.3   | 9.9      | 21.6              |
| 11 <sub>a</sub> | 21 <sup>a</sup>  | 190    | 176             | 130    | 182      | 290             | 17  | 175    | 8.7      | 8.8    | 5.1      | 9.5               |
| 12 <sub>a</sub> | 189              | 19     | 165             | 133    | 288      | 316             | 10.7  | 111    | 6.3      | 5.1    | 5.1      | 7.2               |
| 12 <sub>b</sub> | 214              | 275    | 1.8             | 194    | 296      | 35 <sup>a</sup> | 8.5   | 175    | 1.6      | 8.3    | 5.3      | 6.1               |
| 12 <sub>c</sub> | 141              | 137    | 1.2             | 138    | 261      | 285             | 8.9   | 118    | 8.4      | 8.5    | 6.1      | 3.0               |
| 18 <sub>a</sub> | 118              | 165    | 168             | 167    | 287      | 297             | 10.5  | 148    | 12.9     | 13.1   | 10.0     | 7.3               |
| 18 <sub>b</sub> | 13               | 147    | 122             | 113    | 246      | 249             | 8.5   | 130    | 6.7      | 6.0    | 3.5      | 1.2               |
| 18 <sub>c</sub> | 128              | 153    | 158             | 161    | 238      | 244             | 7.3   | 5.2    | 7.2      | 5.8    | 2.6      | 3.2               |
| 18 <sub>d</sub> | 125              | 163    | 177             | 159    | 269      | 290             | 11.1  | 147    | 14.1     | 9.8    | 4.5      | 5.7               |
| 18 <sub>e</sub> | 161              | 165    | 121             | 120    | 204      | 188             | 13.8  | 165    | 8.2      | 8.1    | 1.6      | 4.3               |
| 18 <sub>f</sub> | 171              | 196    | 157             | 147    | 251      | 249             | 19.0  | 20.2   | 9.0      | 11.5   | 5.7      | 6.0               |
| 19 <sub>a</sub> | 181              | 180    | 146             | 150    | 245      | 293             | 19.8  | 22.2   | 9.6      | 9.5    | 7.0      | 9.0               |
| 19 <sub>b</sub> | 175              | 230    | 172             | 156    | 275      | 328             | 19.0  | 20.1   | 13.1     | 10.7   | 5.5      | 9.0               |
| mean            | 160              | 176    | 167             | 162    | 258      | 283             | 13.1  | 15.6   | 10.1     | 11.0   | 6.1      | 8.9               |
| s.d.            | 36               | 37     | 32              | 36     | 36       | 13              | 4.6   | 5.7    | 4.5      | 6.3    | 2.4      | 7.3               |
| error of mean   | 8.9              | 9.2    | 8.0             | 8.9    | 9.0      | 10.6            | 1.15  | 1.41   | 1.11     | 1.57   | 0.61     | 1.82              |

<sup>a</sup>) Bones with corroded nail

Table 18 Values noted for 60 day old fractures

| animal No | wt of bone ash in mg |        |          |        |          |        | Sr <sup>85</sup> activity in % of dose $\times 10^3$ |        |          |        |          |                  |
|-----------|----------------------|--------|----------|--------|----------|--------|--|--------|----------|--------|----------|------------------|
|           | A part               |        | B part   |        | C part   |        | A part   |        | B part   |        | C part   |                  |
|           | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed   | nailed | unnailed | nailed | unnailed | nailed           |
| 116       | 160                  | 152    | 146      | 140    | 234      | 316    | 87   | 108    | 95       | 81     | 62       | 146 <sup>a</sup> |
| 117       | 147                  | 195    | 133      | 130    | 303      | 267    | 127  | 150    | 68       | 56     | 61       | 67               |
| 118       | 183                  | 180    | 175      | 144    | 248      | 280    | 106  | 121    | 72       | 54     | 50       | 59               |
| 119       | 150                  | 119    | 150      | 143    | 276      | 292    | 50   | 91     | 64       | 64     | 47       | 114              |
| 128       | 152                  | 162    | 192      | 164    | 206      | 267    | 87   | 124    | 156      | 123    | 55       | 79 <sup>a</sup>  |
| 130       | 191                  | 192    | 231      | 199    | 362      | 456    | 61   | 106    | 105      | 108    | 64       | 101 <sup>a</sup> |
| 131       | 239                  | 227    | 192      | 181    | 270      | 293    | 183  | 170    | 88       | 85     | 70       | 109              |
| 16        | 163                  | 161    | 115      | 97     | 206      | 226    | 160  | 155    | 42       | 41     | 57       | 57               |
| 217       | 170                  | 149    | 116      | 89     | 194      | 203    | 230  | 200    | 83       | 50     | 76       | 79               |
| 218       | 105                  | 120    | 85       | 98     | 189      | 202    | 101  | 95     | 33       | 43     | 50       | 17               |
| 251       | 145                  | 183    | 107      | 99     | 225      | 222    | 220  | 281    | 103      | 73     | 92       | 100              |
| 252       | 141                  | 140    | 126      | 101    | 210      | 225    | 81   | 81     | 41       | 35     | 38       | 38               |
| 253       | 120                  | 172    | 133      | 156    | 359      | 354    | 65   | 100    | 75       | 68     | 58       | 58               |
| 254       | 126                  | 149    | 125      | 135    | 313      | 321    | 30   | 54     | 35       | 44     | 33       | 41               |
| 255       | 104                  | 116    | 112      | 113    | 239      | 245    | 67   | 68     | 41       | 37     | 32       | 36               |
| 280       | 177                  | 179    | 149      | 114    | 274      | 280    | 163  | 166    | 57       | 77     | 71       | 100              |
| 281       | 144                  | 162    | 133      | 138    | 235      | 269    | 204  | 220    | 72       | 76     | 80       | 75               |
| 282       | 157                  | 161    | 124      | 108    | 253      | 289    | 122  | 150    | 55       | 83     | 58       | 54               |
| mean      | 154                  | 164    | 141      | 131    | 255      | 278    | 119  | 136    | 71       | 67     | 59       | 76               |
| s d       | 32                   | 27     | 36       | 31     | 52       | 61     | 61   | 58     | 31       | 24     | 16       | 31               |
| error     |                      |        |          |        |          |        |  |        |          |        |          |                  |
| of mean   | 7.6                  | 6.3    | 8.5      | 7.4    | 12.2     | 14.3   | 1.44   | 1.36   | 0.73     | 0.57   | 0.37     | 0.72             |

<sup>a</sup>) Bones with corrosion of nail

Table 19  $t$  values noted for 70 day old *fracta* es

| animal<br>No | wt of bone a h in mg |        |          |        |          |        | Se <sup>3</sup> activity in $\mu$ of dry $\times 10^3$ |        |          |        |          |        |
|--------------|----------------------|--------|----------|--------|----------|--------|--|--------|----------|--------|----------|--------|
|              | A part               |        | B part   |        | C part   |        | A part   |        | B part   |        | C part   |        |
|              | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed   | nailed | unnailed | nailed | unnailed | nailed |
| 132          | 173                  | 190    | 155      | 159    | 271      | 309    | 47   | 81     | 53       | 76     | 36       | 53     |
| 133          | 186                  | 01     | 193      | 00     | 291      | 376    | 112  | 152    | 115      | 107    | 79       | 93     |
| 134          | 174                  | 176    | 182      | 185    | 37       | 382    | 59   | 92     | 88       | 85     | 67       | 75     |
| 135          | 148                  | 144    | 159      | 189    | 318      | 371    | 61   | 81     | 88       | 116    | 71       | 109    |
| 140          | 226                  | 206    | 130      | 121    | 57       | 213    | 19   | 108    | 56       | 63     | 58       | 76     |
| 141          | 191                  | 17     | 148      | 166    | 277      | 260    | 124  | 108    | 54       | 6      | 50       | 10     |
| 144          | 36                   | 220    | 173      | 169    | 72       | 344    | 139  | 111    | 54       | 65     | 56       | 101    |
| 143          | 180                  | 135    | 145      | 146    | 286      | 200    | 103  | 127    | 46       | 11     | 10       | 10     |
| 256          | 9                    | 189    | 110      | 130    | 227      | 211    | 20   | 62     | 138      | 87     | 9        | 103    |
| 257          | 15                   | 139    | 118      | 177    | 260      | 301    | 160  | 158    | 71       | 56     | 15       | 101    |
| 258          | 150                  | 152    | 111      | 100    | 14       | 221    | 173  | 92     | 19       | 42     | 80       | 60     |
| 259          | 141                  | 161    | 103      | 109    | 219      | 210    | 182  | 108    | 09       | 51     | 70       | 58     |
| 260          | 190                  | 118    | 146      | 131    | 208      | 230    | 218  | 237    | 1        | 82     | 86       | 80     |
| 262          | 161                  | 168    | 152      | 119    | 211      | 268    | 81   |        | 51       | 19     | 12       | 11     |
| 263          | 173                  | 181    | 113      | 131    | 230      | 208    | 50   | 50     | 3        | 6      | 28       | 32     |
| 264          | 203                  | 21     | 129      | 118    | 224      | 26     | 132  | 148    | 43       | 37     | 42       | 55     |
| 283          | 165                  | 166    | 170      | 138    | 211      | 200    | 146  | 101    | 00       | 71     | 10       | 10     |
| mean         | 175                  | 188    | 141      | 144    | 260      | 276    | 128  | 138    | 60       | 66     | 10       | 70     |
| s d          | 03                   | 30     | 01       | 30     | 30       | 51     | 61   | 57     | 28       | 1      | 19       | 25     |
| error        |                      |        |          |        |          |        |  |        |          |        |          |        |
| mean         | 55                   | 73     | 58       | 73     | 78       | 104    | 118  | 138    | 667      | 060    | 016      | 010    |

Table 18 *I values noted for 60 day old fractures*

| animal<br>No | wt of bone ash in mg |        |          |        |          |        | Sr <sup>85</sup> activity in % of dose $\times 10^3$ |        |          |        |          |                  |
|--------------|----------------------|--------|----------|--------|----------|--------|--|--------|----------|--------|----------|------------------|
|              | A part               |        | B part   |        | C part   |        | A part   |        | B part   |        | C part   |                  |
|              | unnailed             | nailed | unnailed | nailed | unnailed | nailed | unnailed   | nailed | unnailed | nailed | unnailed | nailed           |
| 116          | 160                  | 152    | 146      | 140    | 234      | 316    | 87   | 108    | 95       | 81     | 62       | 146 <sup>4</sup> |
| 117          | 147                  | 195    | 133      | 130    | 303      | 267    | 127  | 150    | 68       | 56     | 61       | 67               |
| 118          | 183                  | 180    | 175      | 141    | 248      | 280    | 106  | 121    | 72       | 54     | 50       | 59               |
| 119          | 150                  | 149    | 150      | 143    | 276      | 292    | 50   | 91     | 64       | 64     | 47       | 114              |
| 128          | 152                  | 162    | 192      | 161    | 206      | 267    | 87   | 124    | 156      | 123    | 55       | 79 <sup>4</sup>  |
| 130          | 191                  | 192    | 231      | 199    | 362      | 456    | 61   | 106    | 105      | 108    | 64       | 101 <sup>4</sup> |
| 131          | 239                  | 227    | 192      | 181    | 270      | 293    | 183  | 170    | 88       | 85     | 70       | 109              |
| 246          | 163                  | 161    | 115      | 97     | 206      | 226    | 160  | 155    | 42       | 41     | 57       | 57               |
| 247          | 170                  | 149    | 116      | 89     | 191      | 203    | 230  | 200    | 83       | 50     | 76       | 79               |
| 248          | 105                  | 120    | 85       | 98     | 189      | 202    | 101  | 95     | 33       | 43     | 50       | 47               |
| 251          | 145                  | 183    | 107      | 99     | 225      | 222    | 220  | 281    | 103      | 73     | 92       | 100              |
| 252          | 141                  | 140    | 126      | 101    | 210      | 225    | 81   | 81     | 41       | 35     | 38       | 38               |
| 253          | 120                  | 172    | 133      | 156    | 359      | 354    | 65   | 100    | 75       | 68     | 58       | 58               |
| 254          | 126                  | 149    | 125      | 135    | 313      | 321    | 30   | 54     | 35       | 41     | 33       | 41               |
| 255          | 104                  | 116    | 112      | 113    | 239      | 245    | 67   | 68     | 41       | 37     | 32       | 36               |
| 260          | 177                  | 179    | 149      | 114    | 274      | 280    | 163  | 166    | 57       | 77     | 71       | 100              |
| 281          | 144                  | 162    | 133      | 138    | 235      | 269    | 204  | 220    | 72       | 76     | 80       | 75               |
| 282          | 157                  | 161    | 121      | 108    | 253      | 289    | 122  | 150    | 55       | 83     | 58       | 54               |
| mean         | 154                  | 164    | 141      | 131    | 255      | 278    | 119  | 136    | 71       | 67     | 59       | 76               |
| s d          | 32                   | 27     | 36       | 31     | 52       | 61     | 61   | 58     | 31       | 24     | 16       | 31               |
| error        |                      |        |          |        |          |        |  |        |          |        |          |                  |
| of mean      | 7.6                  | 6.3    | 8.5      | 7.1    | 12.2     | 14.3   | 1.44   | 1.36   | 0.73     | 0.57   | 0.37     | 0.72             |

<sup>4</sup>) Bones with corrosion of nail



Table 91. *Values noted for 100 day old fractures*

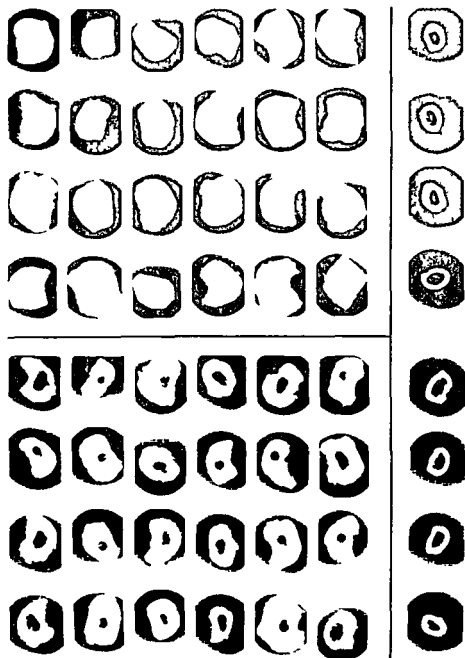
| animal No | wt of bone ash in mg. |        |          |        |          |        | Sr <sup>90</sup> activity in %, of dose $\times 10$ |        |          |        |          |        |
|-----------|-----------------------|--------|----------|--------|----------|--------|---|--------|----------|--------|----------|--------|
|           | A part                |        | B part   |        | C part   |        | A part  |        | B part   |        | C part   |        |
|           | unnailed              | nailed | unnailed | nailed | unnailed | nailed | unnailed  | nailed | unnailed | nailed | unnailed | nailed |
| 168       | 255                   | 988    | 147      | 151    | 270      | 276    | 115   | 115    | 40       | 45     | 1        | 59     |
| 170       | 208                   | 961    | 151      | 156    | 227      | 227    | 83  | 150    | 54       | 53     | 11       | 50     |
| 171       | 170                   | 176    | 133      | 126    | 231      | 300    | 1   | 54     | 33       | 17     | 38       | 50     |
| 172       | 162                   | 156    | 111      | 98     | 241      | 218    | 15  | 54     | 26       | 17     | 36       | 39     |
| 173       | 207                   | 172    | 137      | 148    | 239      | 278    | 9   | 47     | 20       | 25     | 26       | 30     |
| 174       | 164                   | 150    | 114      | 98     | 199      | 187    | 79  | 45     | 93       | 20     | 7        | 27     |
| 175       | 198                   | 197    | 139      | 137    | 252      | 246    | 74  | 91     | 39       | 99     | 34       | 34     |
| 176       | 164                   | 199    | 134      | 140    | 246      | 277    | 28  | 31     | 18       | 18     | 22       | 28     |
| 177       | 195                   | 958    | 133      | 198    | 394      | 312    | 94  | 110    | 35       | 40     | 15       | 54     |
| 178       | 184                   | 169    | 133      | 147    | 270      | 278    | 152   | 144    | 70       | 77     | 80       | 89     |
| 179       | 235                   | 237    | 145      | 190    | 311      | 334    | 68  | 68     | 30       | 18     | 51       | 43     |
| 180       | 197                   | 234    | 137      | 149    | 291      | 239    | 121   | 134    | 51       | 40     | 11       | 12     |
| 181       | 127                   | 192    | 159      | 150    | 334      | 311    | 31  | 19     | 35       | 31     | 32       | 30     |
| 182       | 185                   | 215    | 177      | 160    | 374      | 364    | 63  | 88     | 58       | 59     | 60       | 71     |
| 183       | 231                   | 200    | 161      | 179    | 329      | 376    | 171   | 164    | 61       | 56     | 67       | 81     |
| 220       | 116                   | 126    | 114      | 104    | 994      | 307    | 26  | 37     | 99       | 91     | 17       | 46     |
| 294       | 202                   | 201    | 16       | 132    | 303      | 321    | 48  | 71     | 30       | 34     | 8        | 37     |
| 225       | 117                   | 159    | 121      | 120    | 289      | 293    | 37  | 14     | 27       | 93     | 40       | 37     |
| 226       | 123                   | 152    | 100      | 98     | 23       | 253    | 91  | 108    | 36       | 16     | 54       | 57     |
| 297       | 190                   | 154    | 161      | 136    | 372      | 274    | 40  | 92     | 27       | 13     | 29       | 48     |
| 228       | 139                   | 186    | 103      | 136    | 280      | 354    | 89  | 74     | 38       | 93     | 42       | 34     |
| 229       | 165                   | 911    | 107      | 97     | 238      | 275    | 97  | 80     | 31       | 31     | 41       | 54     |
| 276       | 169                   | 151    | 139      | 121    | 390      | 409    | 17  | 11     | 18       | 21     | 33       | 36     |
| 277       | 142                   | 138    | 140      | 118    | 279      | 280    | 96  | 99     | 91       | 23     | 23       | 27     |
| mean      | 177                   | 190    | 136      | 131    | 285      | 291    | 71  | 81     | 35       | 1      | 11       | 16     |
| s.d.      | 38                    | 42     | 21       | 25     | 52       | 51     | 41  | 39     | 14       | 15     | 14       | 17     |
| error     |                       |        |          |        |          |        |   |        |          |        |          |        |
| of mean   | 77                    | 87     | 42       | 50     | 105      | 105    | 083   | 080    | 099      | 031    | 098      | 034    |

\*) Bones with corrosion of nail

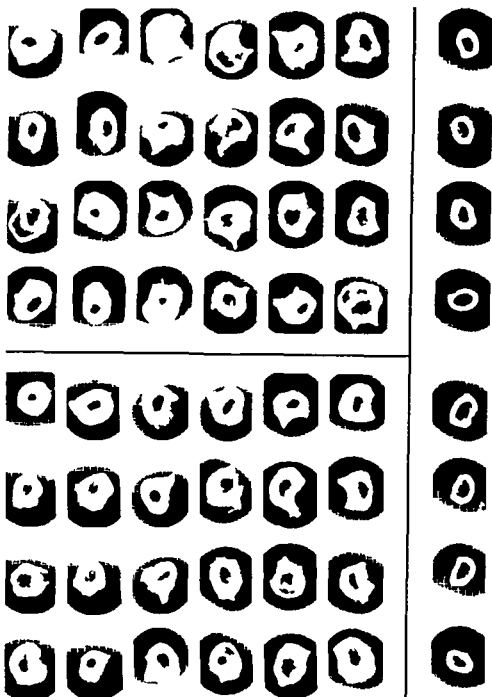
Table 20 Values noted for 120 day old fractures

| animal<br>No     | wt of bone ash in mg |       |          |       |          |       | Sr <sup>88</sup> activity in % of dose $\times 10^3$ |       |          |       |          |       |
|------------------|----------------------|-------|----------|-------|----------|-------|--|-------|----------|-------|----------|-------|
|                  | A part               |       | B part   |       | C part   |       | A part   |       | B part   |       | C part   |       |
|                  | unnailed             | naild | unnailed | naild | unnailed | naild | unnailed   | naild | unnailed | naild | unnailed | naild |
| 148              | 170                  | 164   | 111      | 175   | 298      | 339   | 131  | 104   | 41       | 56    | 56       | 68    |
| 149              | 162                  | 202   | 156      | 183   | 38       | 374   | 18   | 29    | 24       | 27    | 21       | 27    |
| 150              | 229                  | 265   | 141      | 159   | 376      | 351   | 80   | 92    | 23       | 28    | 38       | 14    |
| 151              | 154                  | 163   | 150      | 138   | 289      | 308   | 34   | 57    | 34       | 32    | 33       | 47    |
| 156              | 285                  | 214   | 157      | 190   | 292      | 396   | 84   | 71    | 32       | 45    | 36       | 69    |
| 157              | 227                  | 262   | 133      | 194   | 311      | 316   | 55   | 71    | 21       | 31    | 35       | 42    |
| 158              | 189                  | 236   | 164      | 175   | 311      | 32    | 38   | 44    | 22       | 28    | 20       | 21    |
| 159              | 293                  | 266   | 148      | 206   | 307      | 330   | 40   | 45    | 19       | 22    | 21       | 21    |
| 16               | 187                  | 171   | 153      | 145   | 255      | 289   | 11   | 50    | 12       | 42    | 35       | 44    |
| 166              | 154                  | 149   | 123      | 115   | 258      | 274   | 58   | 82    | 51       | 50    | 63       | 80    |
| 167              | 125                  | 113   | 110      | 116   | 318      | 313   | 26   | 40    | 19       | 21    | 33       | 37    |
| 210              | 152                  | 189   | 132      | 140   | 355      | 376   | 153  | 150   | 106      | 83    | 71       | 124   |
| 211              | 170                  | 187   | 9        | 130   | 299      | 290   | 206  | 191   | 15       | 64    | 80       | 111   |
| 219              | 119                  | 152   | 119      | 119   | 265      | 260   | 79   | 101   | 45       | 43    | 67       | 67    |
| 213              | 123                  | 139   | 122      | 121   | 261      | 269   | 89   | 97    | 55       | 59    | 77       | 92    |
| 214              | 178                  | 164   | 152      | 131   | 265      | 284   | 40   | 38    | 23       | 27    | 25       | 33    |
| 215              | 162                  | 159   | 119      | 138   | 238      | 269   | 46   | 42    | 20       | 25    | 36       | 374   |
| 216              | 191                  | 119   | 141      | 121   | 377      | 195   | 57   | 79    | 60       | 59    | 93       | 77    |
| 218              | 125                  | 155   | 97       | 132   | 312      | 317   | 31   | 59    | 30       | 40    | 32       | 45    |
| 219              | 199                  | 140   | 103      | 108   | 279      | 287   | 35   | 35    | 16       | 21    | 27       | 33    |
| 220              | 144                  | 134   | 99       | 106   | 238      | 280   | 46   | 44    | 23       | 30    | 25       | 42    |
| 221              | 115                  | 149   | 117      | 107   | 296      | 273   | 59   | 71    | 33       | 33    | 30       | 38    |
| 278              | 196                  | 210   | 136      | 143   | 290      | 298   | 104  | 107   | 56       | 69    | 74       | 85    |
| 279              | 173                  | 188   | 144      | 119   | 274      | 273   | 74   | 74    | 37       | 28    | 46       | 54    |
| mean             | 170                  | 181   | 130      | 140   | 293      | 301   | 67   | 74    | 37       | 40    | 45       | 56    |
| s.d.             | 42                   | 4     | 21       | 29    | 98       | 11    | 44   | 38    | 18       | 16    | 22       | 27    |
| error<br>of mean | 8.6                  | 8.5   | 4.3      | 6.0   | 5.8      | 8.4   | 0.90   | 0.78  | 0.38     | 0.33  | 0.45     | 0.56  |

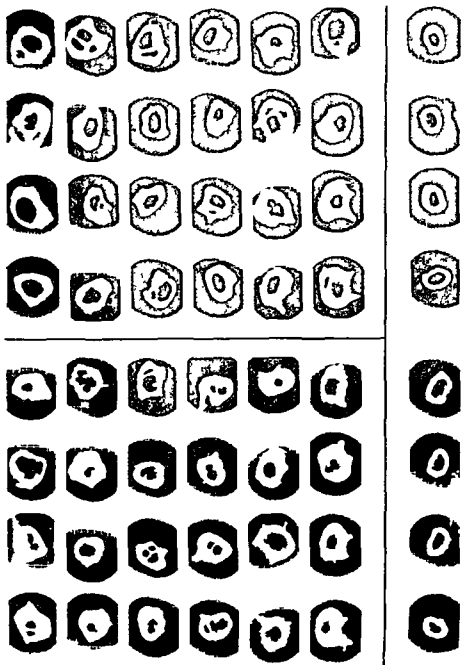
\*) Bones with corrosion of nail



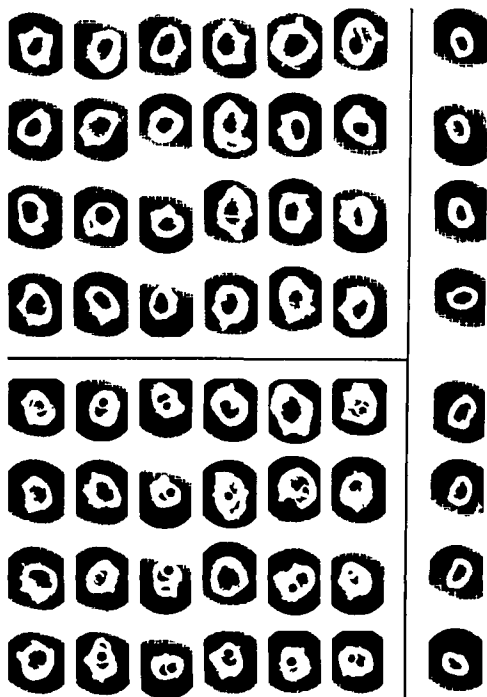
Cross-sectional area of callus in 0 day old fractures Above unnailed Below nailed  
Row to extreme right normal radius



Cross-sectional area of callus in 50 day old fractures Above unnailed Below nailed  
Row to extreme right normal radius



Cross-sectional area of callus in 10 day old fractures. Above unnailed Below nailed  
Row to extreme right normal radius.



Cross sectional area of callus in 120 day old fractures Above unnailed Below nailed  
Row to extreme right normal radius

## REFERENCES

- BAST T H SULLIVAN W E GEIST F O The repair of bone *Anat Record* 31 33 1935
- BALER, G C H Influence of earlier fracture on bone repair in rats studied by means of radiocalcium. *Acta Orthop Scand* 22 85 1952
- Bone salt metabolism in bone growth and bone repair studied in rats by means of  $\text{Ca}^{45}$   $\text{P}^{32}$  and  $\text{Na}^{22}$  *Acta Orthop Scand* 23 247 1954a.
- The importance of bone growth as a factor in the redistribution of bone salt *J Bone & Joint Surg* 36 A 315 1954b
- Rate of bone salt formation in a healing fracture determined in rats by means of radiocalcium *Acta Orthop Scand* 23 169 1954c
- Long term redistribution of bone sodium studied in rat by means of  $\text{Na}^{22}$  radioautography *Acta Orthop Scand* 23 197 1954d
- BALER G C H CARLSON A Post fracture bone salt resorption studied in rats *Acta Orthop Scand* 23 83 1955
- BALER, G C H, CARLSON A LINQOLIST B Evaluation of accretion, resorption and exchange reactions in the skeleton. *Kungl Fys iografiska S ikskapets i Lund Forhandlingar* 23 1 1955a.
- A comparative study on the metabolism of  $\text{Sr}^{90}$  and  $\text{Ca}^{45}$  *Acta Physiolog Scand* 35 56 1955b
- BALER, G C H WENDEBERG B External counting of  $\text{Ca}^{45}$  and  $\text{Sr}^{90}$  in studies of localized skeletal lesions in man *J Bone & Joint Surg* 41 B 558 1959
- BECHTOL, C O FERGLON JR A B LAING P G Metals and engineering in bone and joint surgery The Williams & Wilkins Company Baltimore 1959
- BELL, G H Experiments on bone growth. *Annali della Facoltà di Medicina e Chir della Università degli Studi di Perugia* 50 355 1959
- BELL, G H CHAMBER J W DAWSON I M The mechanical and structural properties of bone in rats on a rachitogenic diet. *J Physiology* 106 286 1947
- BELL, G H CUTHBERTSON D P ORR J Strength and size of bone in relation to calcium intake *J Physiology* 100 299 1941
- BELL, G H OLIVE DUNBAR J A GILLESPIE, J IBALL and JEAN OLIVER Effect of sweet pea poisoning on strength of bone and skin *J Physiology* 139 17 1957
- BERG R M KUGELMASS J V Calcification in callus and fracture repair *Ann Surg* 93 1009 1931
- BOHR H H Studies in fracture healing *J Bone & Joint Surg* 31 A 31 1955
- BOHR H H SORENSEN A H Study of fracture healing by means of radioactive tracers *J Bone & Joint Surg* 32 4 56 1950
- BOHLER L Medullary nailing of Kuntzcher The Williams and Wilkins Co Baltimore 1948
- CALIBRIST P SMITH F C The effect of embalming on the compressive strength of a few specimens of compact human bone *Nat Med Research Inst NIH/N 001056 02 MR 51* 1951 (Cited EVANS F G Stresses and strain in bones 1951)
- CARLSON A Metabolism of radiocalcium in relation to calcium intake in young rats *Acta Pharmacolog et Toxicolog Suppl I* 1951
- On the mechanism of the skeletal turnover of lime salt. *Acta Physiolog Scand* 5 100 1952
- CARTIER P H DE BERNARD B LAGRANGE, J Studies on the repair of fractures using  $\text{P}^{32}$  *Bone structure and metabolism Ciba Foundation* 1956
- COFF D H GREENBERG D M Studies on bone fracture healing. *J Nutrition* 29 61 1945
- COLSTON SIFFIT M. Histological study of the callus of experimental fractures treated with medullary nailing *Mém Acad. Chir Paris* 78 46 1952

- DOWNS JR. W G MCKEOWN R M Histology of healing fractures in rats on normal diet  
*Arch Surg* 25 94 1932
- ESKELUND V MUNK PLUM C Exp investigations into the healing of fractures *Acta Orthop Scand* 19 433 1950
- EVANS F G Stress and strain in bones Charles C Thomas Publisher Springfield Illinois U.S.A. 1957
- Relations between the microscopic structure and tensile strength of human bone  
*Acta Anat* 35 285 1958
- FITTS JR W T ROBERTS B SPOONT S J RITTER, V W The effect of medullary nailing on the healing of fractures An animal study *Sur Gyn Obst* 89 609 1949
- FONTAINE, R CHEVALLIER A MENDEL P WIST E Étude expérimentale histologique et biochimique de la réparation des fractures chez le chien *Lyon Chir* 45 145 1950
- FONTAINE, R MULLER J V SIBILLY A Resultat obtenus dans le traitement des fractures et des pseudoarthroses des os longs par l'encolage centromedullaire a ciel ouvert. *Revue de Chir* 92 1 1954
- GALLZI W GIANELLI A Influence of intramedullary nailing on the growth and repair of long bones in the course of development. *Arch Orthop Milano* 6 74 1953
- GRANJON F SOELB R L'encolage centro medullaire des os longs *Revue de Chir Orthopedique* 41 475 1955
- GRIESSMANN H REICH H Vergleichende Untersuchungen über Ablauf der Knochenbruchheilung bei Marknagelung und bei der mit Gipsverband behandelte Fracturen  
*Arch Klin. Chir* 205 455 1944
- HASCHKE KLUNDER, R Reaktive feingewebliche Veränderungen bei der Marknagelung  
*Ztschr Orthopedie* 83 120 1952
- HERTZ, J Studies on the healing of fractures E Munksgaard Copenhagen 1936
- HEY GROVES E W Exp principles of the operative treatment of fractures and their clinical application. *Lancet* 1 513 1914
- An experimental study of the operative treatment of fractures *Brit J Surg* 7 211 1920
- HABLER, C REISS O Exp Untersuchungen über die Festigkeit des Knochens im normalen und im Zustand der Bruchheilung *Deutsche Ztschr Chir* 46 760 1936
- JØRGENSEN HJ Experimentelle undersøgelser over fracturhelingen ved osteosyntese  
*Arnold Busch København* 1941
- KAPSHAMMER, G Zur Frage der knorpeligen Callusbildung *Burchows Archiv f Patholog Anat* 152 157 1898
- KARCHER, H Der Ca P Stoffwechsel bei der normalen und gestörten Knochenbruchheilung sowie in frischen und konservierten Transplantaten Ein Nachweis mit den radioaktiven Isotopen  $P^{32}$  und  $Ca^{45}$  *Langenbecks Archiv Klin Chir* 275 1 1952
- KEY J A The effect of a local calcium depot on osteogenesis and healing of fractures  
*J Bone & Joint Surg* 16 176 1934
- KEY J A, ODELL, R T Failure of excess minerals in the diet to accelerate the healing of experimental fractures *J Bone & Joint Surg* 37 1 37 1955
- KUNTSCHEK, G Die Marknagelung von Knochenbrüchen (Tierexp.) *Klin Wchnschr* 1 26 1940
- Callus ohne Knochenbruch. *Zbl. Chir* 68 857 1941
- Die Ursachen der Callusbildung bei der Frakturheilung *Beitr Klin Chir* 191 189 1955
- Das Callusproblem *Arch Orthop Unfallchir* 49 1 1957
- Ein Callusmodell *Zbl Chir* 82 1689 1957
- The Kuntscher method of intramedullary nailing *J Bone & Joint Surg* 40 4 1 1958
- LACROIX, P Discussion of paper by Cartier de Bernard & Lagrange *Ciba Foundation symposium Bone structure and metabolism* London 1956
- LAMBOTTE H Chirurgie Operatoire des Fractures Paris 1913
- LAMBRINUDI C. Intramedullary Kirschner wires in the treatment of fractures *London R Soc Med Proc* 33 153 1940
- LAURITZEN G K. Medullary nailing *Act Chir Scand Suppl* 147 1949



- LEBLOND C P WILKINSON G W BELANGER L F ROBICHOV J Radio autographic visualisation of bone formation in the rat *Amer J Anat* 86 289 1940
- LINDSAY M K HOWES E L The breaking strength of healing fractures *J Bone & Joint Surg* 13 491 1931
- MAATZ R Erfahrungen der Kieler Klinik bei der Marknagelung *Ztschr Orthop* 4 149 1943
- Die Bedeutung des Markraumreizes für die Frakturheilung *Buns Beitrage Kl Chir* 177 363 1943
- MACDONALD N S Kinetic studies of skeletal metabolism by external counting of injected radioisotope *J Laboratory & Clinical Medicine* 52 541 1958
- MACDONALD N S LORICA P C PETRIELLO L J Healing bone fractures and simultaneous administration of radio isotopes of sulfur calcium and yttrium *Am J Physiology* 191 185 1957
- MAJ G TOAJARI E Osservazioni sperimentali sul meccanismo di resistenza del tessuto osseo lamellare compatto alle azioni meccaniche *Chir Org Moderno* 2° 541 1937
- MARSHAK A BYRON JR R L A method for studying healing of bone *J Bone & Joint Surg* 17 95 1945
- MCKEOWN R M LINDSAY M K HARVEY S C HOWES E L The breaking strength of healing fractured fibulae of rats *Arch Surg* 24 458 1932
- MCLEAN F C URIST M R Bone repair in rats with multiple fractures *Am J Surg* 80 685 1940
- Newer knowledge of post fetal osteogenesis *Bulletin of Hospital for Joint Diseases* 14 431 1951
- MATZEN P E Vom Einfluss mechanischen Einwirkungen auf die Kallusbildung *Beitr Klin Chir* 188 97 1954
- MULLER MEERNACH K Die Bolzung der Brüche der langen Röhrenknochen *Zbl Chir urg* 60 1718 1933
- NILSSON U Biophysical investigations of the mineral phase in healing fracture *Acta Orthop Scand Suppl* 37 1959
- PAUWELS L Grundriss einer Biomechanik der Frakturheilung *Ztschr Orthop* 72 62 1946
- PONSETI I V SHEPARD R S Lesions of the skeleton and of other mesodermal tissues in rats fed sweet pea (*Lathyrus Odoratus*) seeds *J Bone & Joint Surg* 36 A 1031 1954
- PRITCHARD J J RUZICKA A J Comparison of fracture repair in the frog lizard and rat. *J Anat* 84 236 1950
- RAISCH O Exp Beiträge zur Frage der Osteosynthesis mit besonderer Berücksichtigung der Marknagelung nach Kuntscher *Beitr Klin Chir* 175 548 1943—44
- RIEDER W SCHUMANN G Unsere Indikation zur Marknagelung der langen Röhrenknochen *Deutsche Ztschr f Chir* 207 415 1943
- ROTH H Knochenmarkveränderungen nach Marknagelung *Schweiz Med Wchnschr* 75 7 1945
- RUHL V RUSH H L A technique for longitudinal pin fixation of certain fractures of the ulna and for the femur *J Bone & Joint Surg* 21 619 1939
- SILANI C ARMANTE S Ricerche sperimentali sul trattamento delle fratture secondo Kuntscher *Arch Orthop Milano* 3 165 1949
- SOEUR R Intramedullary pinning of diaphyse fractures *J Bone & Joint Surg* 28 309 1946
- STREET D M One hundred fractures of the femur treated by means of the diamond shaped medullary nail *J Bone & Joint Surg* 33 A 659 1951
- TRUETA J CAVADIAS A V Vascular changes caused by the Kuntscher type of nailing *J Bone & Joint Surg* 37 B 49 1955
- URIST M R MCLEAN F C Calcification in the callus in healing fractures in normal rat *J Bone & Joint Surg* 23 1 1941
- Control of calcification in the fracture callus in rachitic rats *J Bone & Joint Surg* 3 283 1941

- WEIR J B DE BELL G H CHAMBERS J W Strength and elasticity of bone in rats on rachitogenic diet *J Bone & Joint Surg* 31 B 444 1949
- WENDEBERG B 1961 To be published in *Acta Orthop Scand*
- WERTHEIM M G COT EVANS Stress and Strain in bone 1957
- VOSE G P KUBALA JR A L Bone strength and its relationship to X ray determined ash content *Human Biology* 31 261 1959
- WRAY J B LYNCH C J The vascular response to fracture of the tibia in the rat *J Bone & Joint Surg* 41 A 1143 1959

WISCONSIN  
LIBRARY,

SCIENCE,  
FOR



S M S MEDICAL COLLEGE LIBRARY  
DUE DATE SLIP

*This book is to be returned on or before the date  
marked below —*

A fine of 25 nP will be charged for each day  
the book is kept overtime

|  |  |  |
|--|--|--|
|  |  |  |
|--|--|--|



16 11 19

1

1. 4







ACTA ORTHOPAEDICA SCANDINAVICA  
SUPPLEMENTUM No LI

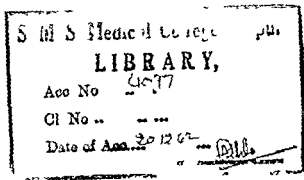
---

FROM THE ORTHOPAEDIC HOSPITAL OF THE INVALID FOUNDATION  
HELSINGFORS HEAD & LÄNGENSKJÖLD MD

# NERVOUS AND VASCULAR INFLUENCE ON LONGITUDINAL GROWTH OF BONE

AN EXPERIMENTAL STUDY ON RABBITS

HENRY TROUPP



---

EJNAR MUNKSGAARD  
COPENHAGEN 1961

PRINTED IN FINLAND BY  
FRENCCKELLSKA TRYCKERI AKTIEBOLAGET  
HELSINGFORS 1961

## ACKNOWLEDGEMENTS

I am very grateful indeed to A. LANGENSKIÖLD M.D. Surgeon in Chief of the Orthopaedic Hospital of the Invalid Foundation for suggesting and supervising this thesis which he did with the utmost care. I should like to express my appreciation of the many long hours he devoted to this task.

I should like to thank K. E. KALLIO M.D. Professor of Orthopaedic and Traumatologic Surgery for kindly permitting this thesis to be presented.

I should also like to thank my esteemed friend and teacher G. AF BJÖRKESTEN M.D. to whom I owe all that I may know of neurological surgery.

I am grateful to V. RITAMA M.D. Chief Pathologist of the Kivela Hospital for his care in checking the histological sections to Miss L. R. ZURNDORFER M.A. (Oxon) for checking the English to Mr. H. LINDROOS for the statistical analysis to Miss L. MIKKOLA for technical assistance and roentgenograms to Miss R. KOSKELA and Miss V. LAURILA for making the histological sections and to Mr. T. SANTAMAKI for the photomicrographs.

This investigation was aided by grants from the Sigrid Juselius Foundation and from Finska Läkaresällskapet which I hereby gratefully acknowledge.



## INTRODUCTION

The shape of a bone is predetermined for the species and the bone in question and independent of the other parts of the organism (*Fell* *Fell & Robinson* *Selby & Murray Selye*) The length (and the girth) of a bone however is modified by hormonal nutritional mechanical nervous and vascular factors

Apparently *Stephen Hales (1727)* was the first to note that longitudinal growth of long bones takes place only at the epiphyses though he did not recognize the importance of his discovery which he mentions only in passing His observation was soon confirmed by *DuHamel* *Haller* and *Hunter* none of them however seems to have understood the role of the epiphysal cartilage plate nor the process of enchondral ossification first mentioned by *Miescher* and described in detail by *H. Muller* *DuHamel* believed that interstitial growth of bone also occurred it is now established that longitudinal growth of long bones occurs almost exclusively at the epiphysal cartilage plate (*Banks & Compere* *Bisgard & Bisgard* *Boerema* *Dubreuil* *Gatewood & Mullen* *Haas 1917a* *Hefferich 1877* *Humphry 1861* *Maass* *Silf erskiold* *Vahlquist* *Wegner*) though there have been a few dissenters (*Hellstadius 1947* *Hellstadius 1951* *Korneu* *Latarjet* *Policard Wolff*)

Gigantism and dwarfism due to pituitary disturbances are a well-known feature in affections of the pituitary and hypothalamus in childhood (*Ford*) dwarfing can be produced experimentally by early hypophysectomy and gigantism by administration of pituitary growth hormone Growth however is also governed by the thyroid the gonads and possibly by the thymus It has been suggested that the pituitary growth hormone need be present in only very small amounts to ensure comparatively normal growth provided there is an adequate supply of thyroid and sex hormones (*Matson*) On the other hand an excess of both thyroid and estrogen caused retardation of growth in the rat (*Su uki*)

Lack of certain nutritional factors such as vitamin D retards longitudinal growth (*Duthie* *Sissons*) as does a quantitatively deficient diet (*Pratt & McCance* *Winters & al*) however longitudinal growth is less



## HISTORICAL REVIEW

### Nervous influence on longitudinal growth of bone

There is some anatomical basis for the assumption that the nervous system has a direct influence on bone growth it is well known that bone marrow contains many nerve fibres which apparently enter it with blood vessels and nerve endings have been observed in close contact with osteoblasts in young animals (*DeCastro*) and in compact bone in adults (*Hurrell Ignatov*) It has even been thought that trophic centres regulating bone growth exist in the spinal cord (*Curcio*) though the experimental method supporting this view is open to criticism

Much experimental work has been done on the effect of denervation on bone growth most investigators have used peripheral nerve section and a few have performed nerve root section A glance at a sketch of the spinal cord and the nerve roots of the lower limb in relation to the sympathetic nervous system (Fig 1) shows that there is a considerable difference between denervation achieved by peripheral nerve section and denervation achieved by nerve root section Section of peripheral nerves produces complete denervation if extensive enough whereas motor or

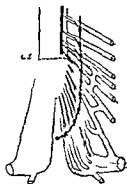


Fig 1 Diagram of sympathetic innervation of lower limb in man redrawn after *Halse & Smithwick*  
Preganglionic fibres ——— Postganglionic fibres

sensory nerve root section abolishes only part of the innervation of a limb even if section of both motor and sensory nerve roots is performed the sympathetic nerve supply remains (Fig 2)

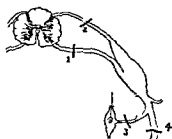


Fig 2 Diagram showing relation of sympathetic chain to spinal cord below the level of L<sub>4</sub> in man.

Preganglionic fibres ——— Postganglionic fibres

1 — section of motor nerve root sympathetic fibres not affected

2 — section of sensory nerve root sympathetic fibres not affected

1 + 2 — section of motor and sensory nerve roots sympathetic fibres not affected.

3 — section of sympathetic postganglionic fibres

4 — section of peripheral nerve sympathetic fibres also severed.

After peripheral nerve section the most general finding has been atrophy of bones with slight retardation of longitudinal growth (*Allison & Brooks Armstrong Bergmann Ghillini Howell Poltorf*) It is however stated by some (*Allison & Brooks Grey & Carr Joseph*) that the atrophy caused by simple immobilization of a limb is as severe as that caused by nerve section Others have found hypertrophy and acceleration of growth after nerve section (*Ghillini Kassowitz Milne Edwards Nasse Ring 1961 Schiff Vulpian*) Vulpian stated that hypertrophy is due to an increased susceptibility to trauma in a limb lacking normal protective sensation and Ghillini suggested that the different results were due to differences in the amount of running about allowed to the experimental animals The denervation experiment which produced hypertrophy of the lower jaw (*Milne Edwards Schiff*) i.e. section of the mandibular nerve was repeated by Ollier and Sulger with negative results they attributed the hypertrophy previously reported to infection

A few claim that denervation has a definite retarding effect on longitudinal growth (*Fischer v Langenbeck Ollier Samuel*) but Fischer merely asserts this Ollier gives no actual measurements Samuel's results apply to regeneration of feathers in doves and v Langenbeck stresses the individual variations in the response to peripheral nerve section



Peripheral nerve injuries in children are reported to cause retardation of growth (*Combes & al Ford Remak*) and in a case of syringomyelia in a child slight shortening of the affected arm was reported (*Bermann*).

Thus at least in experimental work peripheral nerve section seems to have very little effect on longitudinal growth of bone and some (*Borel Friedl & Schin Hert Kapsammer Selje & Bajus*) even go so far as to deny that peripheral nerve section has any influence on longitudinal growth of bone.

There are a few papers dealing with the effect of experimental nerve root section on bone (*Corbin Corbin & Hinsey Eloesser Gillespie Grey & Carr Kurl Ring 1961 Tower*) but none of these experiments have been performed on very young animals. *Corbin Corbin & Hinsey* and *Eloesser* were concerned with the joint changes found after section of sensory nerve roots. *Gillespie* and *Grey & Carr* found atrophy and slight retardation of growth after motor nerve root section but not after sensory root section. *Rings (1961)* results refer to the length of the diaphysis only. *Tower* found atrophy after posterior root section combined with section of the spinal cord above and below the segments subjected to rhizotomy but longitudinal growth seemed unaffected.

*Kure* alone claimed that section of nerve roots had a direct influence on bone but he gave no measurements and his experiments were mainly concerned with the regeneration of the 5th toe in the Japanese giant salamander.

It is interesting that experimental nerve section whether section of peripheral nerves or of nerve roots should have so slight an effect on longitudinal growth of bones in view of the retardation of growth so often seen after paralysis of a lower limb by poliomyelitis. This retardation can be correlated with the degree of paralysis (*Gullickson & al 1950 Humphry 1862 Lindholm Rathiff Ring 1957 Ring 1958a Ring 1958b Stinchfield & al*) but all agree that individual cases show considerable deviation from the statistical mean.

*Ring (1957)* says that paralysis is not the direct cause of retardation of growth and postulates a chronic vascular factor perhaps brought about by muscle loss. *Barr* seems to favour a similar view. Retardation of growth in a limb with congenital deficiency of vasomotor control has been described by *Krepler*. Blood flow in a limb is reported to be decreased by abolished muscle function (*Ollier Sulger*) immobilization (*Hulten*) and denervation (*Kemp & al*). Changes in the pattern and decrease in the size of the blood vessels have been found after section of the sciatic nerve in rabbits (*Ferguson & Akahoshi*). Several investiga-

tions however have given quite contrary results (*Hulth & Olerud Imig & al Schroder & Seyfarth*) and arteriovenous anastomoses have been demonstrated in legs paralyzed by poliomyelitis (*Braibanti Piulachs & Vidal Barraquer*) Furthermore there is no evidence that a cold and clammy limb is particularly likely to develop retardation of growth (*Ratliff*) nor have any consistent changes in peripheral blood flow been found after poliomyelitis (*Abramson & al Dohn McPherson & Kessel Wiggins & al*)

At present therefore evidence on this subject is highly controversial and it should be remembered that the possibility of direct damage to vessel walls by the poliomyelitis virus has been suggested (*Prick*)

Since increased blood flow may lead to overgrowth of a limb (see page 13) sympathectomy might be expected to cause overgrowth Increased blood flow has been demonstrated in muscles after sympathectomy on dogs (*Louenstein & al*) increased growth of auricular hair after cervical sympathectomy on rabbits has been found (*Pye Smith Stirling*) as well as enhanced regeneration after injury to a sympathectomized ear (*Liek*) Stimulation of the sympathetic chain in dogs caused slight retardation of growth of bones (*Gullickson & al 1951*) Increased growth of bones however has generally not been found (*Bacq Bisgard 1931 Bisgard 1933 Cannon & al McCullagh & al Ollier Ring 1961 Simon*) nor was increased growth seen after unilateral sympathectomy on a monkey with bilateral paralysis of the lower limbs after poliomyelitis (*Bisgard 1933*) *Goetz & al Gullickson & al (1951)* and *Kishikawa* are the only ones who have found increased growth of bones after sympathectomy on experimental animals (rabbits and dogs) *Goetz & al* consider earlier failures to be due to the fact that only lumbar sympathectomy was performed which does not cause sympathetic denervation above the knee when they measured the bones of the foot evidence of increased bone growth after lumbar sympathectomy was found This tallies with reports on increased growth of short legs after sympathectomy in man (*Barr Barr & al R I Harris Harris & McDonald Robertson*) as well as with reports on increased growth after the use of sympatholytic drugs (*Kottke & al*) There are however discordant views (*Bombelli Fahey*) and some (*Ogilvie Robertson*) do not think that sympathectomy is justified as a treatment for retardation of growth

The difference between the effect of experimental nerve section and the effect of poliomyelitis may be related to the function of the autonomic nervous system affection of the autonomous nervous system is a well recognized feature in poliomyelitis (*Collins & al Fanconi Hagel*)

stam Kottke & al McPherson & Kessel Lundbaek Moldr er Sabin Sabin & Ward Smith & al Spencer & al Steindler Stenport Zinany & al ) Fanconi claimed that the skin and temperature changes in acute poliomyelitis are caused by lack of movement destruction of lateral horn cells and arterial hypoplasia (Piulachs & Vidal Barraquer Telford & Stopford) He also thought that retardation of growth is caused by a lesion of trophic nerves apparently referring to the observation of DeCastro that nerve fibres can be found in close contact with osteoblasts in young animals On the other hand a sympathetic paralysis should cause vasodilatation whereas the poliomyelitic limb is notoriously cold blue and clammy suggesting overfunction of the vasoconstrictive fibres this vasoconstriction is reported to be absent in the acute stage (Trott & al 1956 Trott & al 1958)

### Vascular influence on longitudinal growth of bone

Since an adequate supply of nutriment is essential for the growing cells of the growth cartilage it is self-evident that circulation is an important factor in the longitudinal growth of bone Many authors (Brodin Silfverskiöld Trueta) have stressed this point but others (H A Harris Pease Wilson & Thompson) have thought functional factors equally important and it has been maintained that stasis or moderate ischaemia have no effect on longitudinal growth (Borel Dickinson Helferich 1887 Grey & Carr)

Experimental research has been mainly directed towards finding measures for increasing bone growth apparently because of the practical value that a reliable procedure for stimulating growth would have

Longitudinal growth of bone has been stimulated experimentally by many methods -- trauma to the medullary cavity (Ferguson Creville & Janes Kishikawa Leander) plugging of the medullary cavity (Carpenter & Dalton Trueta) foreign material near epiphysial plates (Chapchal & Zeldenrust Ford & Canales Pease Wu & Miltner) stripping of periosteum (Brodin Compere & Adams Elo Lacroix Langenskiöld 1957 Ollier Wu & Miltner) arteriovenous fistula (Doerr & Janes Janes & Musgrove Kelly & al ) venous stasis (Bergmann Janes & Musgrove Kishikawa Pearse & Morton 1930 Serelle Wu & Miltner) electrolysis (Wilson & Percy) and heating (Richards & Stofer) Negative reports on the effect of venous stasis (Borel Dickinson Grey & Carr Helferich 1887 ) implantation of foreign material (Bohlman Haas 1938 Herndon & Spencer Meisenbach Montgomery & In

gram) and heating (Ring & Lee) have been published the effect of ultrasound in moderate doses is reported to be nil (Vaughen & Bender) and retardation of growth is found after higher dosage (De Forest & al)

It has been suggested that the common denominator for procedures resulting in stimulation of bone growth is an increase in the blood flow (Janes & Musgrove) though blood flow is decreased immediately distal to an arteriovenous fistula at least at first (Pauporte & al) Arkin & Katz thought that immobilization of the operated limb with consequent decrease of pressure on growth cartilages is an important factor in accelerating growth

A few studies have been concerned with the effect of decreased vascular supply to bones ligation of the femoral artery (Friedl & Schinz Latarjet Milne Edwards Ollier Pearse & Morton 1931 Sousa Pereira) sometimes combined with peripheral nerve section (Borel Friedl & Schinz) or division of the nutrient artery of the bone (Latarjet) The results have generally been insignificant Some shortening at the end of the growth period was seen after plugging of the nutrient canal in new born rabbits (Brookes) and extensive excision of the femoral and popliteal artery caused retarded healing of experimental fractures (Pearse & Morton 1931) Retardation of growth in metacarpal and metatarsal bones after direct stripping of periosteum and perichondrium has been found (Haas 1917b Sousa Pereira) and it is well known that transplanted growth cartilages being deprived of their blood supply lose a considerable part of their growth potential (Aron & Simon Axhausen Brucke Enderlen Haas 1916 Heikel 1939 Heikel 1960b Helferich 1899) the contrary results of Rehn & Wakabayashi stand alone and are perhaps due to some error in the interpretation of the histological observations The effect of interrupting either the epiphysial or the metaphysial blood supply to the epiphysial cartilage plate has been studied in many cases epiphysiodesis was induced with closure of the epiphysial line (Trueta & Amato)

These methods which led to retardation of growth all imply direct handling of the growth cartilage and however gentle these methods may have been the possibility that the direct trauma had some effect cannot be excluded

There seem to be no studies concerned with the effect of temporary ischaemia on growing limbs it is stated that the time limit for operating in a bloodless field in the limbs is 2 hours (Bunnell) and this time limit can be extended (Mason & Bell) In the dog experimental studies have demonstrated neuromuscular functional impairment and delayed return of the normal temperature of the skin and muscles after

ischaemia of more than 4 hours duration (*Piletta & al Walker & al* )  
 Since growth processes are particularly active in the growth cartilage  
 which is thought to be very sensitive to disturbances in nutrition the  
 effect of temporary ischaemia on the growth cartilage seemed worth  
 studying

gram) and heating (*Ring & Lee*) have been published the effect of ultrasound in moderate doses is reported to be nil (*Vaughen & Bender*) and retardation of growth is found after higher dosage (*De Forest & al*)

It has been suggested that the common denominator for procedures resulting in stimulation of bone growth is an increase in the blood flow (*Janes & Musgrove*) though blood flow is decreased immediately distal to an arteriovenous fistula at least at first (*Pauporte & al*) *Arkin & Kat.* thought that immobilization of the operated limb with consequent decrease of pressure on growth cartilages is an important factor in accelerating growth

A few studies have been concerned with the effect of decreased vascular supply to bones ligation of the femoral artery (*Friedl & Schinz Lalarjet Milne Edwards Ollier Pearse & Morton 1931 Sousa Pereira*) sometimes combined with peripheral nerve section (*Borel Friedl & Schinz*) or division of the nutrient artery of the bone (*Lalarjet*) The results have generally been insignificant Some shortening at the end of the growth period was seen after plugging of the nutrient canal in new born rabbits (*Brookes*) and extensive excision of the femoral and popliteal artery caused retarded healing of experimental fractures (*Pearse & Morton 1931*) Retardation of growth in metacarpal and metatarsal bones after direct stripping of periosteum and perichondrium has been found (*Haas 1917b Sousa Pereira*) and it is well known that transplanted growth cartilages being deprived of their blood supply lose a considerable part of their growth potential (*Aron & Simon Axhausen Brucke Enderlen Haas 1916 Heikel 1959 Heikel 1960b Helferich 1899*) the contrary results of *Rehn & Wakabayashi* stand alone and are perhaps due to some error in the interpretation of the histological observations The effect of interrupting either the epiphysial or the metaphysial blood supply to the epiphysial cartilage plate has been studied in many cases epiphysiodesis was induced with closure of the epiphysial line (*Trueta & Amato*)

These methods which led to retardation of growth all imply direct handling of the growth cartilage and however gentle these methods may have been the possibility that the direct trauma had some effect cannot be excluded

There seem to be no studies concerned with the effect of temporary ischaemia on growing limbs it is stated that the time limit for operating in a bloodless field in the limbs is 2 hours (*Bunnell*) and this time limit can be extended (*Mason & Bell*) In the dog experimental studies have demonstrated neuromuscular functional impairment and delayed return of the normal temperature of the skin and muscles after

## GENERAL PLANNING OF EXPERIMENTS

Rabbits 13—17 days old were used generally weighing 140—270 grams the comparatively large size of rabbits facilitates early intervention and good roentgenograms their quick growth ensures that any changes produced by experimental procedures show soon

To avoid as far as possible the effect of secondary contractures on longitudinal growth (Bunnell Kritter & Blount Langenbeck Schubert) rather short follow up periods were used

The bones selected for measuring were the femur and the tibia which are the longest bones of the rabbit thus minor errors in measuring were not likely to influence the results unduly Since most of the operations performed turned out to have caused some contracture at least in the hip symmetrical positioning of the hind limbs for roentgenographic measuring in the live animal was difficult The use of roentgenographic measurements was therefore rejected and the femur and the tibia were measured with calipers when the animals had been killed and the bones removed and cleaned this method of measurement is exact to 0.5 mm (Greville & Jones) The length of the femur was measured from the upper articular surface of the femoral head to the articular surface of the medial condyle the length of the tibia was measured from the eminentia intercondylica to the measure between the two articular surfaces of the lower end of the tibia All measurements were corrected to the nearest 0.5 mm

As the denervation procedures were found to be technically easier on the left side the left hind limb was used as the experimental limb throughout the investigation the right hind limb serving as a control Autopsy was performed on all animals subjected to section of nerve roots to check which roots had been severed

The bones studied were preserved in formalin they were usually decalcified in a 10 per cent solution of the disodium salt of diethylenetetraacetic acid and stained with haematoxylin — van Gieson

## DENERVATION

### Anatomy

The hind limb of the rabbit is innervated mainly by the nerve roots L6—L7—S1—S2 (*Goetz & al Krause*) of which L7 and S1 are generally the thickest ones. In the rabbit the spinal cord extends well into the sacral canal thus the nerve roots are short and easily identifiable.

### Technique

Under local anaesthesia with lidocaine the lumbar spine was exposed through a dorsal midline incision. General anaesthesia with ether was then induced and a suitable low lumbar hemilaminectomy was performed with some unroofing of the sacral canal. The nerve roots were exposed, identified and cut. Penicillin was introduced into the wound which was then closed in two layers with catgut.

In the course of preliminary experiments not reported upon here it was found both at operation and at autopsy that there were usually two nerve roots thicker than the others; these nerve roots were on the whole identifiable at autopsy as L7 and S1 though in some cases pre- or postfixation was found. In the animals with a four root section the two thick roots were cut as well as one root cranial and another caudal to them. In the animals with a three root section the two thick nerve roots were cut and one root caudal to them. In one group of two root sections the more caudal of the two thick nerve roots and the one caudal to this were cut; in another group of two root sections the more cranial of the two thick nerve roots and the one immediately cranial to this were cut.

Useful data were obtained from 45 animals.

### Section of 4 motor nerve roots

After section of 4 motor nerve roots complete or almost complete paralysis of the denervated limb was observed in all animals; no animal



LENGTH OF LUMBAR AND TIBIA IN THE RABBIT  
after section of  
4 lumbosacral motor nerve roots

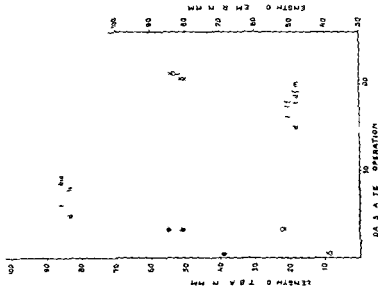


Fig 3 Indices (1-2) denote different rabbits killed at the same number of days after operation

LENGTH OF LUMBAR AND TIBIA IN THE RABBIT  
after section of  
3 lumbosacral motor nerve roots

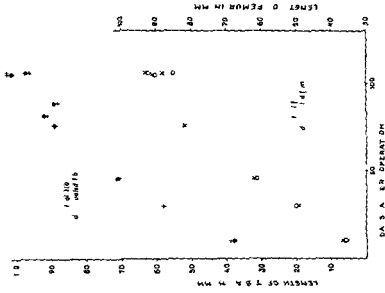


Fig 4 Indices (1-2) denote different rabbits killed at the same number of days after operation

was able to use the denervated limb although in some instances there was flexion and adduction in the hip. There was severe atrophy of the muscles of the denervated limb after 4 weeks.

The effect of section of 4 lumbosacral motor nerve roots on the length of the femur and the tibia is shown in Fig. 3.

### Section of 3 motor nerve roots

After section of 3 motor nerve roots paralysis almost as severe as occurred after section of 4 motor nerve roots was observed in the denervated limb; no animal was able to use the denervated limb. In the muscles atrophy of the same degree as occurred in the group with section of 4 motor nerve roots was found.

The effect of section of 3 lumbosacral motor nerve roots on the length of the femur and the tibia is shown in Fig. 4.

### Section of 2 motor nerve roots

Section of 2 motor nerve roots was performed in 8 animals. Two variants of this — see paragraph on technique page 18 — were used each on 4 animals. However, as no difference was found between these two groups either on postoperative examination, roentgenological examination, or by measurement, they are reported upon as a single group.

In all animals weakness of the denervated limb was found immediately after operation, but all animals were able to use the denervated limb well, possibly because of overlapping innervation.

The effect of section of 2 motor nerve roots on the length of the femur and the tibia is shown in Fig. 5.

### Section of 4 motor and 4 sensory nerve roots

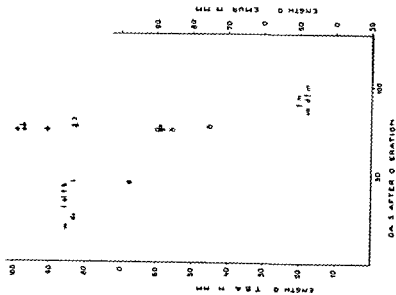
After section of 4 motor and 4 sensory lumbosacral nerve roots complete or almost complete paralysis of the denervated hind limb was found as in the group subjected to section of 4 motor nerve roots. No animal was able to use the denervated limb.

The effect of section of 4 motor and 4 sensory lumbosacral nerve roots on the length of the femur and the tibia is shown in Fig. 6.

### Section of 4 sensory nerve roots

In the postoperative examinations of the animals subjected to sensory denervation it was found that they retained a crude motility in the

LENGTH OF I I MUR AND TIBIA IN THE RABBIT  
after section of  
2 lumbosacral motor nerve roots



LENGTH OF I I MUI AND TIBIA IN THE RABBIT  
after section of  
4 motor and 4 sensory lumbosacral nerve roots

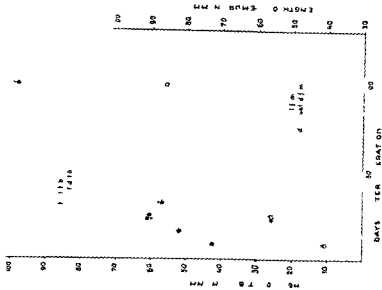


Fig 5 Indices (1-5) denote different rabbits with the same number of days after operation

denervated limb (*Lassek & Moyer*) though they could not move the limb in a purposeful manner. When lifted by the nape of the neck they would wriggle violently, licking with both legs and stretching them forward, but the denervated limb would then sink down, apparently owing to lack of positional sense.

At autopsy subluxation of the hip was found in some cases. Muscle atrophy was much less severe than in limbs subjected to motor denervation. Any grazing was generally found around the ankles, as a rule bilaterally.

The effect of section of 4 sensory nerve roots on the length of the femur and the tibia is shown in Fig. 7. There was considerable retardation of growth of the denervated femur, but not of the tibia. In all animals surviving operation for 25 days or more, considerable thickening of the lower epiphysis of the femur was observed, with distortion of the epiphyseal line. The roentgenological and histological findings (see page 24 and 30) suggest that the retardation of growth was due to the rabbit's inadvertently injuring an anaesthetic limb. This assumption seems to tally with the experimental production of Charcot joints by sensory denervation (*Eloesser*) and with the bone changes associated with syringomyelia in children (*Ford*). In no case was any sign of purulent arthritis found in the knee joint at autopsy.

To prove the assumption that retardation of growth of the femur found after sensory denervation was due to traumatization of an anaesthetic limb, experiments were performed with the denervated limb protected. Insertion of the denervated and skinned limb under the abdominal skin was considered a suitable method. Five animals were subjected to sensory denervation at 14 days of age; a fortnight later the denervated limb was skinned and inserted under the abdominal skin. At this stage, check roentgenograms showed no appreciable changes in the bones of the denervated limbs. The results of this two-stage procedure are shown in Fig. 8. Growth in the femur was much less retarded than in the series subjected to sensory denervation only, and in the tibia acceleration of growth was even seen.

### Roentgenological observations

After section of 4 motor nerve roots, osteoporosis of the bones of the denervated limb (Fig. 9) was seen in all but two of the animals; of these two, one died 2 days after operation and one was killed 16 days after operation. Another animal killed 16 days after operation already showed slight osteoporosis.

LENGTH OF LUMBAR AND TIBIAL NERVES IN THE RABBIT  
after section of  
4 lumbosacral sensory nerve roots

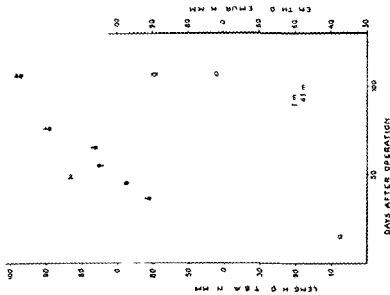


Fig. 7 indicates (1-2) denote different rabbits killed the same number of days after operation

LENGTH OF LUMBAR AND TIBIAL NERVES IN THE RABBIT  
after section of 4 lumbosacral sensory nerve roots  
and inversion of the denervated limb under the abdominal skin

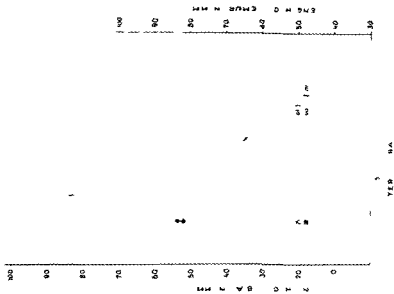


Fig. 8 indicates (1-2) denote different rabbits killed the same number of days after operation and inversion of the denervated limb under the abdominal skin was performed 14 days after denervation (see page 60)

In the group subjected to section of 3 motor nerve roots definite osteoporosis was seen from the 30th day after operation and one animal had already shown osteoporosis in the denervated limb 9 days after operation (Fig 10)

In the group subjected to section of 2 motor nerve roots osteoporosis was observed in the denervated limb in all animals despite the very slight and transient changes in the function of the limb (Fig 11)

After section of 4 motor and 4 sensory nerve roots osteoporosis was observed in the denervated limb in all but one of the animals this animal died 8 days after operation

As mentioned previously retardation of growth and thickening was found in the denervated femur of the rabbits subjected to section of 4 sensory nerve roots Fifteen days after operation an area of decreased density was seen in the distal epiphysis of the denervated femur and the proximal epiphysis of the denervated tibia (Fig 12) 25 days after operation there was distortion and thickening of the distal end of the femur and the epiphysial line appeared widened and blurred with spurs of bony overgrowth at the margins (Fig 13) Thirty six days after operation the roentgenological findings were similar to those observed 25 days after operation and in addition the proximal end of the denervated tibia was somewhat thicker than it was in the control limb as was the cortex of the bone In the animals killed 45 55 65 76 and 106 days after operation these changes had progressed with considerable stunting of the longitudinal growth of the femur and thickening of the upper end of the tibia From the 45th day after operation it was seen that the structure in the upper end of the femur had become progressively more irregular (Fig 14) This may possibly be explained by the subluxation found in some cases

These roentgenological observations suggest that deformity and retardation of longitudinal growth of the femur was due to inadvertent traumatization of an anaesthetic limb Roentgenological examination of the animals in the control series in which the denervated limb was skinned and tucked under the abdominal skin 14 days after denervation showed much fewer changes Roentgenograms of these animals immediately before the second operation showed no definite changes roentgenograms of the 2 animals that died 24 days after denervation (10 days after the second procedure) showed some decrease in density in the lower femoral epiphysis in the animal that died 45 days after denervation there was thickening of the lower femoral epiphysis the trabeculae of which seemed coarse (Fig 15) the trabecular arrangement in the upper end of the femur

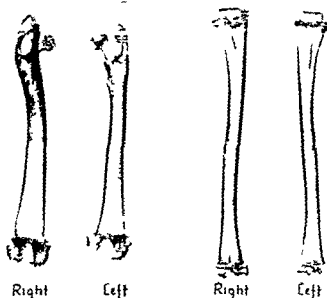


Fig 9 Femora and tibiae of rabbit subjected to section of 4 lumbosacral motor nerve roots on the left at 17 days of age and killed 103 days after operation. Left femur 4 mm shorter than right. Left tibia 2 mm shorter than right. Considerable osteoporosis of left femur and tibia.

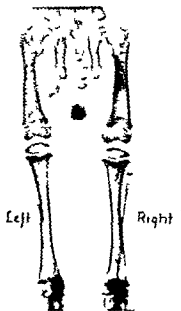


Fig 10 Femora and tibiae of rabbit subjected to section of 3 lumbosacral motor nerve roots on the left at 15 days of age. roentgenogram of live animal 9 days after operation. Osteoporosis of left femur and tibia.

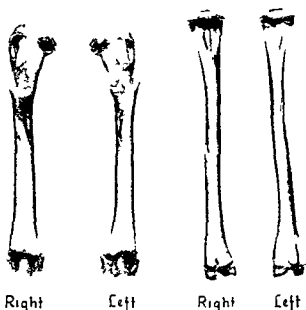


Fig 11 Femora and tibiae of rabbit subjected to section of 2 sacral motor nerve roots on the left at 15 days of age and killed 75 days after operation. Left femur 0.5 mm shorter than right tibiae of equal length. Slight osteoporosis of left femur and tibia.

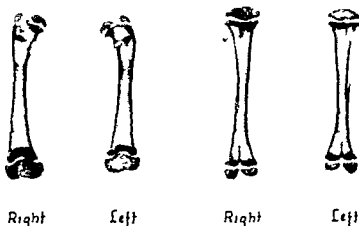


Fig 12 Femora and tibiae of rabbit subjected to section of 4 lumbosacral sensory nerve roots on the left at 15 days of age and killed 15 days after operation. Osteoporosis of lower femoral and upper tibial epiphysis.



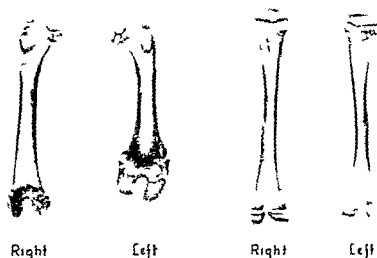


Fig 13 Femora and tibiae of rabbit subjected to section of 4 lumbosacral sensory nerve roots on the left at 15 days of age and killed 25 days after operation. Left femur 6 mm shorter than right. Left tibia 2.5 mm shorter than right. Thickening of lower femoral epiphysis. epiphyseal line blurred. bony overgrowth at lateral margins. Osteoporosis in lower femoral and upper tibial epiphysis. Compare Fig 1 page 9.

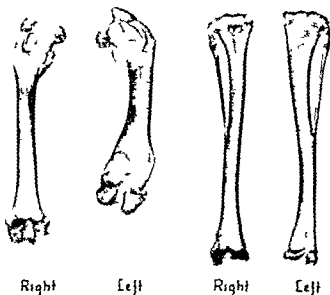


Fig 14 Femora and tibiae of rabbit subjected to section of 4 lumbosacral sensory nerve roots on the left at 14 days of age and killed 106 days after operation. Left femur 17 mm shorter than right. Left tibia 2 mm longer than right. Thickening and distortion of left femur. slight thickening of upper end of tibia.

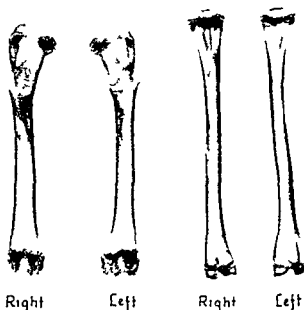


Fig 11 Femora and tibiae of rabbit subjected to section of 2 sacral motor nerve roots on the left at 15 days of age and killed 75 days after operation. Left femur 0.5 mm shorter than right tibiae of equal length. Slight osteoporosis of left femur and tibia.

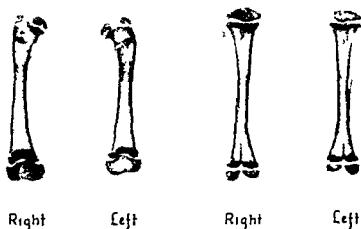


Fig 12 Femora and tibiae of rabbit subjected to section of 4 lumbosacral sensory nerve roots on the left at 15 days of age and killed 15 days after operation. Osteoporosis of lower femoral and upper tibial epiphysis.



Fig 17 a Section of lower end of femur in rabbit subjected to severance of 4 sensory nerve roots on the left at 15 days of age and killed 25 days after operation Transverse fracture in metaphysis ( $\downarrow$ ) delayed ossification of growth cartilage ( $\rightarrow$ )  $\times 35$



Fig 17 b Magnification of square marked on Fig 17 a Crack in metaphysis at top ( $\downarrow$ ) growth cartilage at bottom ( $\rightarrow$ )  $\times 25$

was also disturbed as was that of the upper tibial epiphysis. In the animal killed 70 days after denervation there was also thickening of the femur, in particular of the lower epiphysis but the femora were equal in length and the trabecular arrangement in the denervated femur (Fig. 16) was by no means as disturbed as in the case of the animal killed 76 days after denervation without insertion of the limb under the abdominal skin.

### Histological observations

Only in the groups subjected to sensory denervation were changes found that could be called pathological; other groups are not worth reporting on.

In the rabbit killed 15 days after severance of 4 sensory nerve roots slight disarrangement of the cellular columns in the lower femoral growth cartilage was found in some sections; otherwise the histological findings in this animal were within normal limits.

In the rabbit killed 25 days after section of 4 sensory nerve roots a transverse fracture of the metaphysis of the lower end of the denervated femur was found; there was some disarrangement of the growth cartilage and it seemed probable that the fracture had affected the growth cartilage (Fig. 17). Such a fracture provides a natural explanation for the retardation of growth observed after sensory denervation; it also explains the thickening.

A similar fracture was observed in the animals killed 36, 55 and 65 days after operation; in the other animals only very slight disarrangement of the cell columns in the growth cartilage was found and it is doubtful whether such changes could be called pathological.

The animals that had had the denervated limb inserted under the abdominal skin showed no pathological changes in the growth cartilages.

## DEVASCULARIZATION

### Anatomy

The hind limb of the rabbit is mainly fed through the femoral artery (*Brookes & Harrison Krause*). Arteries branching off from this are the medial circumflex artery immediately below the inguinal groove and the lateral circumflex artery a little further down which in turn gives rise to the nutrient artery of the femur. The line of devascularization was generally distal to this latter artery.

### Technique

Since several investigators had found that ligation of the femoral artery did not appreciably affect longitudinal growth (*Friedl & Schin. Lатарjet Milne Edwards Ollier Pearse & Morton 1930 Sousa Pereira*) presumably because of the quick development of collateral blood supply a more drastic method of devascularization seemed necessary to retard growth. A further consideration was the avoidance of direct trauma to the growth cartilages.

The following procedure was evolved.

A transverse skin incision was made distal to the left groin under local anaesthesia with lidocaine. The lateral femoral muscles were cut in the mid thigh, the sciatic nerve was identified and the muscles posterior to it were severed. Next the medial femoral muscles were cut, the femoral vessels were freed from the femoral nerve, tied and severed. It was then ascertained that no remnants of muscle posterior to the femur remained undivided. In the first few animals of the series the femoral nerve was sometimes accidentally severed. Penicillin was introduced into the wound and the skin was closed with catgut. Thus the femur, the femoral and the sciatic nerves and the skin were left intact whereas all vessels and muscles in the thigh were cut. By means of this procedure the blood supply of the tibia was severely reduced and since the multiplying cells of the growth cartilage are apparently nourished from the epiphysial side (*Trueta & Amato Trueta & Little Trueta & Morgan*) the lower femoral growth cartilage also suffered ischaemia.

# LENGTH OF FEMUR AND TIBIA IN THE RABBIT after subtotal devascularization

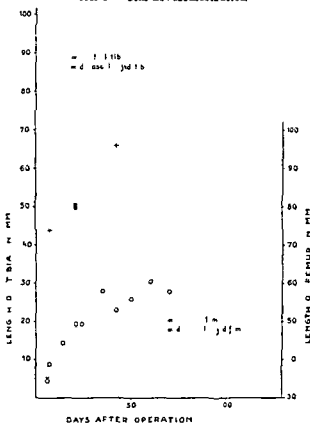


Fig 18 Indices (1 2) denote different rabbits killed the same number of days after operation.

To produce temporary ischaemia a piece of thin rubber tubing was tied tightly around the left thigh of the rabbit for 2—7 hours. Only the tibiae were studied since the femur of the rabbit is well protected by muscles the rubber tubing had to be applied just above the knee and it is possible that blood was still being supplied to the lower femoral epiphysis. These animals were killed 1 4 7 14 and 40—42 days after the period of ischaemia.

All the animals were killed by decapitation immediately afterwards a barium sulphate suspension was injected into the thoracic aorta. No attempt was made to measure the pressure of the injection since perfectly adequate angiograms can be obtained without such a device (*Morgan*). Roentgenograms were then taken of the lower part of the animal in the prone and supine positions the hind limbs were skinned

# LENGTH OF FEMUR AND TIBIA IN THE RABBIT immediately after subtotal devascularization

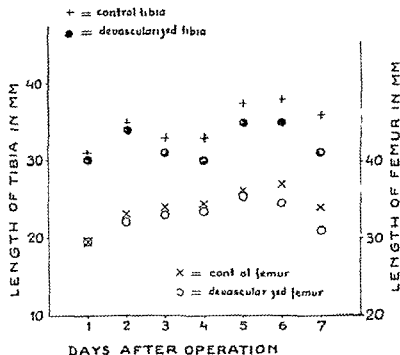


Fig 19

and exarticulated at the hip and new roentgenograms taken of the skinned limbs

The growth cartilages affected by operation were subjected to histological examination however the distal tibial growth cartilage was examined in a few instances only

Useful data were obtained from 87 animals

## Subtotal devascularization

Little active motility remained in the devascularized limbs which were mostly held in a position of flexion adduction and internal rotation at the hip flexion at the knee and extension at the ankle In animals surviving operation for more than 21 days the upper articular surface of the tibia was distorted and cup-shaped

The effect of subtotal devascularization on longitudinal growth of the

# LENGTH OF FEMUR AND TIBIA IN THE RABBIT after subtotal devascularization

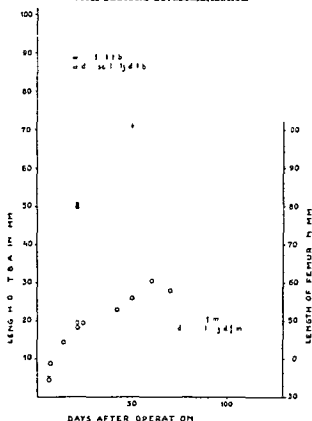


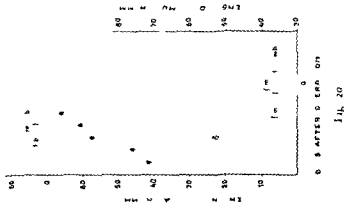
Fig 18 Indices(1 2) denote different rabbits killed the same number of days after operation

To produce temporary ischaemia a piece of thin rubber tubing was tied tightly around the left thigh of the rabbit for 2—7 hours. Only the tibiae were studied since the femur of the rabbit is well protected by muscles the rubber tubing had to be applied just above the knee and it is possible that blood was still being supplied to the lower femoral epiphysis. These animals were killed 1 4 7 14 and 40—42 days after the period of ischaemia.

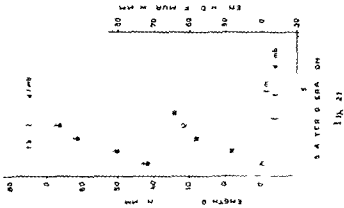
All the animals were killed by decapitation immediately afterwards a barium sulphate suspension was injected into the thoracic aorta. No attempt was made to measure the pressure of the injection since perfectly adequate angiograms can be obtained without such a device (*Morgan*). Roentgenograms were then taken of the lower part of the animal in the prone and supine positions the hind limbs were skinned



# LENGTH OF HUMER AND TIBIA IN THE RABBIT after severing of muscles in the mid thigh



# LENGTH OF HUMER AND TIBIA IN THE RABBIT after severing of femoral vessels



The sample mean of the difference in length between the control and experimental tibiae is 25.4 per cent in the group (x) subjected to subtotal devascularization and lumbar sympathectomy and 21.8 per cent in the group (y) subjected to subtotal devascularization only. The variance is 49.4 and 75.9 respectively. If the sample means are assumed to be normally distributed and if it is assumed that sympathectomy would increase longitudinal growth by approximately 2 per cent during the time these animals lived after sympathectomy (Goet & al.) the null hypothesis is  $m_y - m_x = 2$ . Then the variable  $z = \bar{y} - \bar{x} - 2$  is normally distributed with the mean value = 0 and the variance  $\sigma_z^2 = \frac{1}{8} (49.4 + 75.9)$  i.e. the standard deviation is 4.0.

The probability of a deviation of  $z \leq -5.6$  is  $P(z \leq -5.6) = P(\frac{z}{4.0} \leq -1.4) = 1 - \Phi(1.4)$  or  $P(z \leq -5.6) = 0.08$ . The null hypothesis can thus be rejected with a probability of 92 per cent.

TABLE 1. Difference in length of tibiae of rabbit after subtotal devascularization of left hind limb and left lumbar sympathectomy

| Animal | Length of<br>right tibia | left tibia | Difference<br>as percentage of control limb |
|--------|--------------------------|------------|---|
| 111    | 71 mm                    | 53 mm      | -5  |
| 121    | 68                       | 51.5       | 24  |
| 122    | 74                       | 55         | 26  |
| 141    | 66                       | 48         | 27  |
| 142    | 63                       | 47         | 25  |
| 151    | 58                       | 46         | -1  |
| 153    | 57                       | 34         | 40  |
| 161    | 62                       | 53         | 15  |

Difference in length of tibiae of rabbit  
after subtotal devascularization of left hind limb

| Animal | Length of<br>right tibia | left tibia | Difference<br>as percentage of control limb |
|--------|--------------------------|------------|---|
| 112    | 73 mm                    | 58 mm      | 21  |
| 113    | 73                       | 59         | 19  |
| 123    | 70                       | 40.5       | 42  |
| 124    | 73.5                     | 59         | 20  |
| 131    | 78                       | 6          | 21  |
| 13     | 75                       | 59         | -1  |
| 15     | 67                       | 56         | 18  |
| 162    | 69                       | 61         | 12  |

All animals were killed 41 or 42 days after devascularization (27 or 28 days after sympathectomy). In the numbering the first two digits denote the litter the last digit the individual.

## Temporary ischaemia

The effect of increasingly long periods of ischaemia on longitudinal growth of the tibia of the rabbit is shown in Fig. 22. It will be seen that ischaemia lasting up to 5 hours caused no significant retardation of growth though changes in the other tissues of the limb were apparent. These changes tally well with the observations of *Paletta & al.* since even 6 weeks after the period of ischaemia the animals were not able to use the injured limb normally.

The postmortal angiograms (see *Roentgenological observations* page 43) make it clear that even short term ischaemia produces changes in the peripheral vessels of the limb since it was impossible to differentiate between the effect of temporary ischaemia and the effect of the vascular injury: thus line of investigation was abandoned.

RETARDATION OF GROWTH OF TIBIA IN THE RABBIT  
seen 40—42 days after 2—7 hours of ischaemia  
(given as percentage of length of control limb)

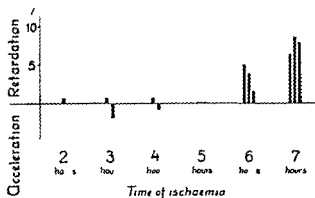


Fig. 22. Each column represents one rabbit.

## Roentgenological observations

Widening of the lower femoral and both tibial epiphyseal lines was seen 24 hours after subtotal devascularization (Fig. 23). This widening persisted throughout the first postoperative week; it gradually diminished and narrowing of the lower femoral epiphyseal line was already found 9 days after operation. Fourteen days after devascularization narrowing of all epiphyseal lines was seen (Fig. 24); narrowing was also seen 21, 35, 42, 50, 60 and 70 days after operation (Fig. 25). In many cases a streak of

The sample mean of the difference in length between the control and experimental tibiae is 25.4 per cent in the group (x) subjected to subtotal devascularization and lumbar sympathectomy and 21.8 per cent in the group (y) subjected to subtotal devascularization only. The variance is 49.4 and 75.9 respectively. If the sample means are assumed to be normally distributed and if it is assumed that sympathectomy would increase longitudinal growth by approximately 2 per cent during the time these animals lived after sympathectomy (Goetz & al.) the null hypothesis is  $m_y - m_x = 2$ . Then the variable  $z = \bar{y} - \bar{x} - 2$  is normally distributed with the mean value = 0 and the variance  $\sigma_z^2 = \frac{1}{8} (49.4 + 75.9)$  i.e. the standard deviation is 4.0.

The probability of a deviation of  $z \leq -5.6$  is  $P(z \leq -5.6) = P(\frac{z}{4.0} \leq -1.4) = 1 - \Phi(1.4)$  or  $P(z \leq -5.6) = 0.08$ . The null hypothesis can thus be rejected with a probability of 92 per cent.

TABLE 1. Difference in length of tibiae of rabbit after subtotal devascularization of left hind limb and left lumbar sympathectomy

| Animal | Length of<br>right tibia | left tibia | Difference<br>as percentage of control limb |
|--------|--------------------------|------------|---|
| 111    | 71 mm                    | 53 mm      | 25  |
| 121    | 68                       | 51.5       | 24  |
| 122    | 74                       | 55         | 26  |
| 141    | 66                       | 48         | 27  |
| 142    | 63                       | 47         | 25  |
| 151    | 58                       | 46         | 21  |
| 153    | 57                       | 34         | 40  |
| 161    | 62                       | 53         | 15  |

Difference in length of tibiae of rabbit  
after subtotal devascularization of left hind limb

| Animal | Length of<br>right tibia | left tibia | Difference<br>as percentage of control limb |
|--------|--------------------------|------------|---|
| 11     | 73 mm                    | 58 mm      | 21  |
| 113    | 73                       | 59         | 19  |
| 13     | 70                       | 40.5       | 42  |
| 14     | 73.5                     | 59         | 0   |
| 131    | 78                       | 62         | 21  |
| 13     | 75                       | 59         | 21  |
| 15     | 67                       | 56         | 18  |
| 16     | 69                       | 61         | 12  |

All animals were killed 41 or 42 days after devascularization ( 7 or 28 days after sympathectomy). In the numbering the first two digits denote the litter the last digit the individual.

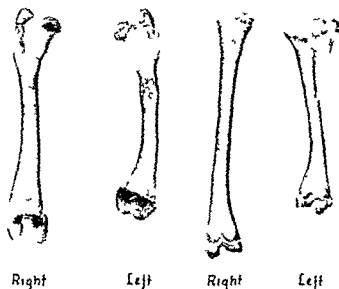


Fig 25 Femora and tibiae of rabbit subjected to subtotal devascularization of left hind limb at 15 days of age and killed 70 days after operation. Left femur 10 mm shorter than right; left tibia 14.5 mm shorter than right. Left femur slightly bent into varus distortion of left upper tibial articular surface; streak of bony tissue crossing epiphyseal line; narrowing of lower epiphyseal line of left femur and of both epiphyseal lines of left tibia.



Fig 6 Tibiae of rabbit subjected to temporary ischaemia of left leg for 4 hours at 14 days of age and killed 24 hours after release of constricting band. Widening of both epiphyseal lines of left tibia. Compare histological findings Fig 4r and Fig 4.

bony tissue crossing the epiphyseal line from the epiphysis to the metaphysis was seen (Fig 25) histological examination corroborated this. In almost all cases overgrowth of the fibula in relation to the tibia was observed this was probably due to the bony central epiphysiodesis always found in the tibia after subtotal devascularization but not in the fibula (see *Histological observations* page 54)

In the control group subjected to division of the femoral muscles with preservation of the femoral vessels there was periosteal reaction in the femur at the site of operation just as in the devascularized limbs (Fig 24) and some osteoporosis of the femur and tibia but in the epiphyseal lines no changes were seen. The osteoporosis was attributable to inactivity since the rabbits used the injured limb much less than the control limb.

In the control group subjected to section of the femoral vessels no recognizable changes were seen in plain roentgenograms not even in the case of the animal killed 35 days after operation in which the tibia of the operated limb was 2 mm shorter than that of the control limb.

Temporary ischaemia of 2 hours duration had caused no changes visible in plain roentgenograms 24 hours after the release of the rubber band. 4 days after the period of ischaemia there was slight osteoporosis in the upper tibial epiphysis possibly due to the inactivity of the limb.

Three hours of ischaemia had caused some widening of the upper tibial epiphyseal line 24 hours after the release of the rubber band. Four days after the period of ischaemia this was no longer seen though some swelling of cells in the growth cartilage was found at histological examination (see page 53). Forty and 41 days after operation there was some osteoporosis in the upper tibial epiphysis and periosteal reaction in the femur where the rubber band had been.

Four hours of ischaemia had caused widening of both tibial epiphyseal lines 24 hours after the release of the rubber band (Fig 26) and slight widening was seen 4 days after the period of ischaemia. Seven days after the procedure no widening of the epiphyseal lines was seen but osteoporosis of the upper tibial epiphysis was observed. Fourteen days after the period of ischaemia there was no difference between the experimental and the control limb. 40 and 41 days after ischaemia there was osteoporosis of the upper tibial epiphysis and periosteal reaction in the femur where the rubber band had been.

Five hours of ischaemia had caused widening of the tibial epiphyseal lines 24 hours after the release of the rubber band widening was also seen 4 days after the period of ischaemia. There was no widening of the epiphyseal lines 7 days after the procedure but some osteoporosis of the

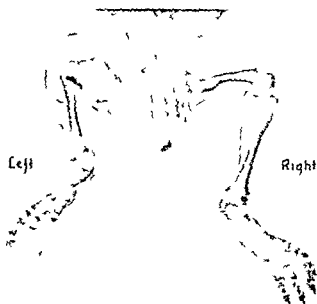


Fig 27 Postmortem angiogram of lower part of rabbit subjected to subtotal devascularization of left hind limb at 15 days of age and killed 72 hours after operation. Considerable revascularization in operated limb compare roentgenogram of skinned limb (Fig 28)



Fig 28 Lateral roentgenogram of skinned left hind limb of rabbit subjected to subtotal devascularization at 15 days of age and killed 72 hours after operation. Line of division of femoral muscles shows well (4) considerable revascularization of muscles of leg Compare Fig 27

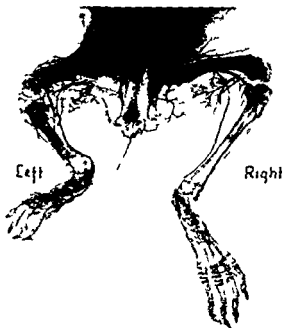


Fig. 9 Postmortal angiogram of lower part of rabbit subjected to subtotal devascularization of left hind limb at 17 days of age and killed 14 days after operation. Increased vascularity in left thigh distal vessels in operated limb thick and tortuous

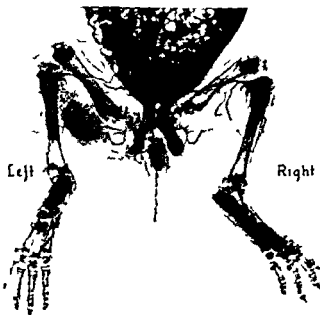


Fig. 30 Postmortal angiogram of lower part of rabbit subjected to temporary ischaemia of left leg for 1 hour at 14 days of age and killed 24 hours after release of constricting band. Impaired filling of vessels in left leg



upper tibial epiphysis was seen. Fourteen days after the period of ischaemia there was no widening and 40 days after ischaemia osteoporosis of the upper tibial epiphysis was seen.

Six and 7 hours of ischaemia had caused widening of the epiphyseal lines 24 hours after the procedure which was still seen on the 7th but not on the 14th day after the period of ischaemia. No widening of the epiphyseal lines was seen 40—42 days after ischaemia.

### *Postmortal angiograms*

After subtotal devascularization no filling of vessels distal to the line of division of muscles and vessels was seen on the 1st or 2nd day after operation. On the 3rd postoperative day there was already considerable revascularization with fine vessels in the muscles of the leg and in the foot (Fig. 27 and Fig. 28). In those few cases where only slight or no filling of distal vessels was seen in the operated limb after the 3rd postoperative day it was assumed that changes in the composition of the barium sulphate suspension were responsible since histological examination showed good regeneration.

Fourteen days after devascularization there was intense hypervascularization around the line of incision in the thigh and also around the knee apparently because of collateral vessels. The more peripheral vessels were somewhat tortuous and showed changes in calibre (Fig. 29) reminiscent of those seen after section of peripheral nerves (*Ferguson & Akahoshi, Hulth & Olerud*). The overall peripheral vascularization however seemed fairly good. Postmortal angiograms of animals killed 21, 35, 42, 50, 60 and 70 days after subtotal devascularization showed similar results.

In the control series subjected to division of femoral muscles with preservation of the femoral vessels the peripheral vascularization was intact. There was hypervascularization around the line of incision in the thigh.

In the control series subjected to section of the femoral vessels in the thigh the peripheral vessels filled well through collaterals. The proximal part of the femoral artery did not fill naturally enough.

Temporary ischaemia of 2 hours duration had caused a slight decrease in vascularization in the leg and narrowing of the femoral artery at the site of constriction in the thigh. 24 hours after the release of the rubber band (Fig. 30) four days after the period of ischaemia no changes were seen in the peripheral vessels but there was narrowing of the femoral



Fig. 31 Postmortal angiogram of lower part of rabbit subjected to temporary ischaemia of left leg for 4 hours at 14 days of age and killed 24 hours after release of constricting band  
Impaired filling of vessels in left leg

artery at the site of constriction a condition also seen 41 days after the period of ischaemia

After 3 hours of ischaemia the peripheral vessels in the injured limb were slightly narrower than in the control limb and the femoral artery was narrow at the site of constriction 24 hours after the release of the rubber band. Four days after the period of ischaemia narrowing of peripheral arteries was still seen but this was not seen in animals killed 7, 14 or 40 and 41 days after the procedure.

Four hours of ischaemia led to a decrease in peripheral vascularization and calibre variation in peripheral vessels seen 24 hours after the release of the rubber band (Fig. 31). This was not seen in an animal killed 4 days after the procedure but in animals killed 7 and 14 days after ischaemia calibre variations were again observed. No changes however were seen 40 and 41 days after ischaemia except narrowing of the femoral artery where the rubber band had been.

Five hours of ischaemia led to decreased peripheral vascularization and irregular filling of main arteries seen 24 hours after the release of the rubber band no pathological changes were seen in the animal killed 4

days after the period of ischaemia but in animals killed 7 and 14 days after the procedure there was increased vascularization around the knee and again irregular filling of peripheral arteries

Six and 7 hours of ischaemia caused similar but more severe changes

However at this point of the investigation it was apparent that a short period of ischaemia i.e. up to 5 hours had caused no significant retardation of growth 6 weeks after the procedure (Fig 22 page 37) The growth cartilages though suffering damage (see page 53) apparently had an astonishing power of regeneration more so than other tissues (skin nerves muscles) in the limb which remained permanently damaged after a period of ischaemia lasting 4 hours or more The angiograms showed that after 2 hours or more of ischaemia definite changes in the vascularization of the injured limb had occurred persisting up to 14 days after the injury Consequently no differentiation could be made between the effects of temporary ischaemia and the effects of more permanent vascular damage It seemed that the difference in effect between long periods of ischaemia and subtotal devascularization was quantitative rather than qualitative it was therefore thought unnecessary to pursue this line of investigation further

### Histological observations

Twenty four hours after subtotal devascularization the cells in the distal femoral growth cartilage showed signs of severe injury swelling and poor staining of cells and disarrangement of cell columns In the upper tibial growth cartilage changes even more severe were seen (Fig 32) In a few instances a transverse split in the growth cartilage was observed (Fig 32 Fig 33) no similar transverse splits were seen in the control limbs

Forty eight hours after devascularization there were already signs of regeneration and 72 hours after devascularization regeneration was well under way (Fig 34) This tallies well with the angiographic findings (page 43) Regeneration then proceeded at a steady rate more and more new cells appearing towards the centre of the cartilage plate (Fig 35)

A forked growth cartilage was seen in several animals (Fig 36) it resembled the patterns observed by *Heikel (1960b)* in his work on epiphyseal transplantation The cells bordering this wedge shaped piece of bone on the metaphyseal side seemed to be necrotic unossified cartilage

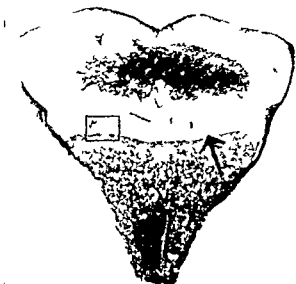


Fig 33 a Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 15 days of age and killed 74 hours after operation. Swelling of growth cartilage. transverse split in cartilage plate ( $\uparrow$ )  $\times 5$

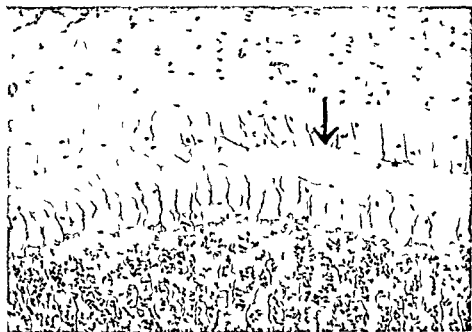


Fig 33 b Magnification of square marked on Fig 33 a. Transverse split clearly shown ( $\downarrow$ )  $\times 70$

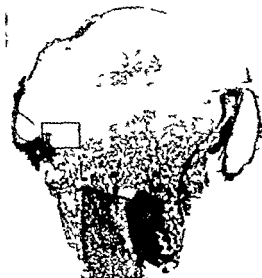


Fig 34 a Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 15 days of age and killed 72 hours after operation. Swelling of growth cartilage  $\times 6$



Fig 34 b Magnification of square marked on Fig 34 a. Regenerating cells near the encoche. necrotic cells at bottom right  $\times 70$



Fig. 35 a Section of lower end of left femur of rabbit subjected to subtotal devascularization of left hind limb at 14 days of age and killed 7 days after operation. Regenerating cells near centre of cartilage plate ( $\downarrow$ )  $\times 5$



Fig. 35 b Magnification of square marked on Fig. 35 a. Regenerating cartilage cells in centre of picture ( $\leftarrow$ ) necrotic cells at top right  $\times 20$

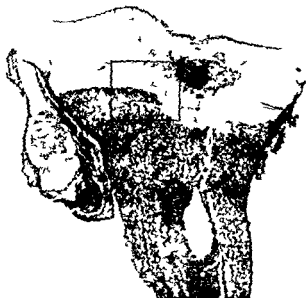


Fig 36 a Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 14 days of age and killed 7 days after operation. Forked growth cartilage formed by regenerating cells growing towards centre of cartilage plate  $\times 6$



Fig 36 b Magnification of square marked on Fig 36 a. Edge of cluster of regenerating cell (\*) necrotic and unossified cartilage cells (†)  $\times 33$



Fig. 37 Section of lower end of left femur of rabbit subjected to subtotal devascularization of left hind limb at 14 days of age and killed 14 days after operation. Two bony bridges uniting epiphysis to metaphysis (↑ ↑)  $\times 7$

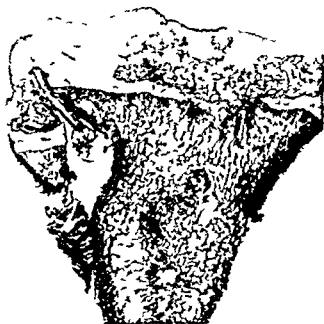


Fig. 38 Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 14 days of age and killed 14 days after operation. Bony bridge uniting epiphysis to metaphysis in centre of growth cartilage (↑)  $\times 6.5$



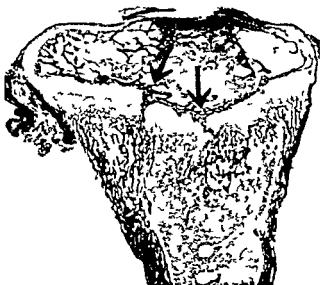


Fig 39 Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 15 days of age and killed 41 days after operation. Growth cartilage well regenerated narrow bony bridges (↓ ↓) uniting epiphysis to metaphysis interference with growth unexpectedly mild (animal 162 page 36)  $\times 7$

Three hours of ischaemia had led to some swelling of cartilage cells 24 hours after the release of the rubber band which tallies well with the widening of the epiphysal line seen in roentgenograms slight changes were still visible 4 days after the period of ischaemia though the roentgenograms were normal by this time

Four hours of ischaemia had caused swelling of cells and disarrangement of cell columns 24 hours after the release of the rubber band (Fig 41) these changes were still visible 4 days after the period of ischaemia but had disappeared by the 7th day

Five 6 and 7 hours of ischaemia caused even more severe changes in the growth cartilage but these persisted for at most a week

At this point these experiments were discontinued for the reasons given in the section on postmortal angiograms (page 45)

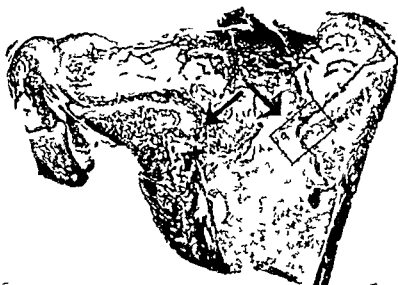


Fig 40 a Section of upper end of left tibia of rabbit subjected to subtotal devascularization of left hind limb at 14 days of age and killed 60 days after operation. Broad bony bridge uniting epiphysis to metaphysis. Arrows (↑ ↓) show interrupted growth cartilage  $\times 4$

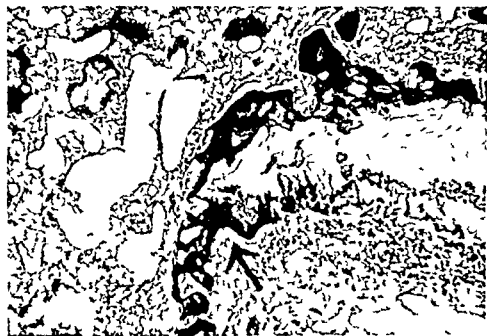


Fig 40 b Magnification of square marked on Fig 40 a. Growth cartilage narrow and (↑ or ↓) apparently nearing end of period of growth 35

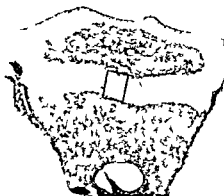


Fig 41 a Section of upper end of left tibia of rabbit subjected to 4 hours of ischaemia at 14 days of age and killed 4 hours after release of constricting band. Swelling of growth cartilage compare Fig 42 a  $\times 5$



Fig 41 b Magnification of square marked on Fig 42 a. Swelling of cartilage cells compare Fig 42 b  $\times 64$



Fig. 42 a Section of upper end of right tibia (control) of rabbit 25 days of age Compare Fig. 41 a  $\times 45$



Fig. 42 b Magnification of square marked on Fig. 42 a Compare Fig. 41 b  $\times 63$

## DISCUSSION

Even after extensive motor denervation of the hind limb of the rabbit retardation of longitudinal growth of the femur and tibia was slight. Section of 4 motor nerve roots caused complete or almost complete paralysis of a hind limb nevertheless the denervated femur was at most 5 per cent shorter than the control femur and the denervated tibia at most 2.5 per cent shorter. Section of 3 motor nerve roots caused almost complete paralysis of the hind limb but the denervated femur was at most 3.5 per cent shorter than the control femur. Section of 2 motor nerve roots (one of the main motor nerve roots was always spared) caused transient paresis but all these animals later used the denervated limb well. Atrophy of muscles and osteoporosis was present in all cases yet no significant retardation of growth was found in either the femur or the tibia. Section of 4 sensory as well as 4 motor nerve roots produced results similar to those observed after section of 4 motor nerve roots.

After paresis caused by poliomyelitis in man growth of a lower limb is often retarded by 5 per cent (i.e. about 4 cm in the average adult). Such a shortening is a handicap and an indication for treatment in order to correct the inequality in length; more severe shortening is not uncommon. The discrepancy between the degree of retardation of growth so often caused by poliomyelitis and the degree of retardation seen after experimental motor nerve root section suggests that paralysis per se leads to only slight retardation; indeed lengthening of the diaphysis after motor nerve root section in puppies has been found (*Ring 1961*).

Admittedly the experimental animals did not live to maturity but by the time a rabbit is 110–120 days old the tibia has reached about 90 per cent of its final length (*Heikel 1960a*) and in these experiments the tibia had tripled its length from the time of rhizotomy. Another difference between retardation of growth caused by motor nerve root section and retardation caused by poliomyelitis is that poliomyelitis generally affects growth in the more distal parts of the limb whereas in my experiments the femur seemed to be more affected than the tibia. However any retarding effect of rhizotomy on the growth of the tibia may have been partially counteracted by the effect of abolition of weight bearing (*Arkin & Katz*).

The role of the vascular disturbances caused by poliomyelitis in producing retardation of growth has been emphasized by many authors (see page 13) and my experimental results support to some degree the theory that when retardation of growth is seen in a paralyzed limb it is caused by interference with its vascular supply. It is not known however whether poliomyelitis causes disturbances in the blood supply through direct injury to vessel walls or through injury to the autonomous nervous system conceivably wasting of paralyzed muscles may contribute to vascular disturbances.

The arteriovenous anastomoses described in limbs paralyzed by poliomyelitis (*Braibanti, Poulachs & Vidal Barraquer*) do not refute this theory of vascular disturbances if it is assumed that the growth cartilages suffer hypoxia since the blood flow in the paralyzed limb is diverted through these arteriovenous shunts. An increase in the longitudinal growth of a paralyzed limb after the surgical creation of an arteriovenous fistula has been reported (*Cooley & al Hierlonn*) but there may be a difference in the effect of one large arteriovenous shunt and that of diffuse arteriovenous anastomoses. The original acceleration of growth in paralyzed limbs (*Lerique Ollier Ring & Ward Seeligmuller*) may be due to the diversion of the blood flow from the paralyzed muscles to the bones before the arteriovenous anastomoses have opened up it may also be due to abolition of weight bearing.

Unexpectedly retardation of growth and thickening of the femora subjected to sensory denervation were observed. The difference in length was 6—21 per cent and some retardation of growth was already seen 15 days after denervation. The tibiae of these animals did not however suffer much nor were they significantly shorter indeed the denervated tibia was longer than the control tibia in some animals.

The short femora were grossly deformed and thickened in many instances and the roentgenological appearance suggested the effect of trauma. This assumption was supported by the discovery of a transverse fracture in the metaphysis of some animals it seemed probable that the fracture had involved the growth cartilage. Such a fracture could easily explain both the deformity and the retardation of growth since an anaesthetic limb is subjected to inadvertent traumatization when the motor supply is preserved. Arthritic changes have been experimentally produced in adult animals through sensory denervation (*Corbin Corbin & Hinsey Eloesser*) and changes similar to those reported here have been observed in patients with syringomyelia (*Ford*) indeed it has been maintained that as long as there is a defect in sensory percep-

tion with the motor supply remaining intact traumatization results (*Potts*)

Insertion of the denervated and skinned limb under the abdominal skin was thought a suitable method for the protection of the denervated limb. Sensory denervation was carried out on a series of 5 animals the denervated limb being inserted under the abdominal skin a fortnight after denervation. There was at most slight deformity of the lower femoral epiphysis and no retardation of growth comparable in severity to that seen after sensory denervation without protective measures. In the protected limb of the animal killed 70 days after denervation considerable lengthening of the tibia was found such lengthening has been observed after the insertion of a sound but skinned forelimb of the rabbit under the skin of the trunk (*W. Muller*). It can thus be considered likely that the changes in the lower end of the femur after sensory denervation were the result of inadvertent traumatization of an anaesthetic limb.

It could not be determined why only the lower end of the femur was affected but since the animals dragged the denervated limb along the floor of the cage the lower end of the femur may have been the part of the limb mostly subjected to trauma. This assumption however was not supported by the fact that grazing when present was generally seen around the ankles.

Subtotal devascularization by severance of muscles and vessels in the thigh caused ischaemia of the limb distal to the line of incision and considerable retardation of growth of the femur and the tibia. The devascularized femur was generally 10–15 per cent shorter and the devascularized tibia some 20 per cent shorter than the femur and tibia of the control limb however there was considerable variation. The apparent shortening of the femur may to some degree have been due to bending of the bone into varus though not all short femora were bent.

It seems probable that longitudinal growth was almost completely arrested during the first week after subtotal devascularization. In the graph showing the retardation of growth 1–7 days after operation (Fig 19 page 33) the control tibia in the animal killed 24 hours after operation is 31 mm long in the animal killed 7 days after operation the control tibia is 36 mm long. At 14 days of age the rabbit tibia grows on an average  $3/4$  of a mm in 24 hours (*Heikel 1960a*) consequently it may be assumed that the tibiae of these two animals were of about the same length at the time of operation and that growth of the devascularized tibia was almost completely arrested during the first week. This tallies well with results seen after epiphyseal transplantation (*Heikel 1960b*)

In postmortal angiograms quick recovery of the severely devascularized limbs was seen the rapid growth of these animals was manifested also in repair after injury and good filling of vessels peripheral to the line of incision in the thigh was already found 72 hours after operation This compares well with studies on vascular regeneration (*Reichert*)

Plain roentgenograms showed widening of the epiphysial line during the first week after operation this widening was mostly attributed to swelling of necrotic cartilage cells but has also been attributed to residual growth of injured cells (*Heikel 1960b*) The epiphysial lines affected by devascularization were all narrower than those in the control limb on the 14th day after operation this tallies well with the histological findings in the present series and with observations of transplanted growth cartilages (*Heikel 1960b*)

Histological examination of the growth cartilages 24 hours after operation showed that the cartilage cells were severely injured but good regeneration was seen 72 hours after operation The quickest regeneration took place at the lateral borders of the growth cartilage it seemed probable that resting cartilage cells survived there and that when revascularization had provided the blood supply they started to multiply pushing inwards to the centre of the cartilage plate On the 7th post-operative day a forked growth cartilage was observed from observations in this and other animals it seemed probable that this double epiphysial line had been formed by regenerating cells pushing inwards to the centre of the growth cartilage leaving the necrotic cells of the primary growth cartilage behind in the metaphysis and that ossification of the necrotic cartilage cells had then been delayed long enough for this forked growth cartilage to become visible The histological findings in my animals were similar to those of *Heikel (1960b)* in his experiments on epiphysial transplantation and the appearance of the wedge-shaped fragment found in some disorders of the growth cartilage (*Langenskiöld 1952 Pylkkänen*) was well illustrated

In some instances a transverse split in the zone of hypertrophied cells was seen during the first week after devascularization no similar split was seen in sections from the control limb Although trauma caused by the cleaning of the bones may have contributed it was thought that these splits represented a pathological weakness in the devascularized growth cartilages since no similar split was seen in the control limb Moreover macroscopic examination showed that the epiphyses in the devascularized limbs were rather loose during the first week after operation



Widening and later slipping of the upper tibial epiphysis in rabbits after ultrasonic injury has been described (*De Forest & al*) In experimental work on epiphysiolsis of the head of the femur in rabbits it has been shown that after direct injury slipping occurs most easily between the zone of hypertrophied cells and the zone of provisional calcification (*Harris & Hobson*) The fact that a transverse split in the growth cartilage may occur after vascular injury as seen in my experiments may have some bearing upon the pathological processes in slipped upper femoral epiphysis particularly as the vascular supply of this epiphysis is easily impaired

From the 9th day after subtotal devascularization a streak of ossifying tissue was seen in the tibia bridging the epiphysis and the metaphysis across the centre of the growth cartilage — i.e. a central bony epiphysiodesis Some kind of epiphysiodesis was found in all devascularized tibiae In many cases two narrow bony bridges were found in the femur the growth cartilage of which is partly divided in two in some instances however the growth cartilage of the femur had regenerated so well that no epiphysiodesis was found Similar bony bridges have been induced by direct experimental surgical trauma (*Campbell & al Ford & Key Haas 1917a Voel Josserand*) after experimental epiphysial fractures a central dip in the growth cartilage has been described (*Brashear*) which may be construed as a minor epiphysiodesis not seriously impeding longitudinal growth

Plain roentgenograms showed a blurred epiphysial line and distortion of the upper end of the tibia in the devascularized limb this was seen 21 days after operation and it was also seen in the animal surviving for 70 days No comparable distortion was ever seen in the femur the lower end of the tibia or the upper end of the fibula The bending of the femur seen in some animals may have been due to different rates of growth of the two condyles though it is not clear why it was always the medial condyle that grew at a slower rate Histological examination of the distorted upper tibial epiphyses always revealed a broad epiphysiodesis a narrow epiphysiodesis as in case 162 (page 36) apparently did not cause distortion It was assumed that the cupped shape of the distorted upper tibial epiphyses was due to hindrance of growth in the centre of the growth cartilage which retained some of its growth potential at the lateral borders Distortion of the articular surface of the epiphysis was not induced in the experiments of *Campbell & al* the epiphysiodesis induced by their methods probably was so complete that growth was arrested Some continuation of growth at the borders of the growth car

tilage seems to be the essential factor that causes the upper articular surface of the tibia to assume a bowl shape. It must be taken into consideration however that the abnormal posture of the devascularized limb may also have played a part.

Similar epiphysiodeses have been found in epiphysial transplantation (Heikel 1960b) and in the course of investigations on the blood supply of the growth cartilage (Frueta & Amato) it seems however that in these cases there was complete inhibition of growth after the formation of a bony bridge between epiphysis and metaphysis. This may be compared to my results in cases 123 and 153 (page 36) in which histological examination revealed that the upper tibial growth cartilage was so severely damaged that only remnants were found. In these cases no bowl shape was seen in the upper tibial epiphysis.

The regular occurrence of these bony bridges between epiphysis and metaphysis at least in the tibia lends some support to the view that regeneration starts from resting cells surviving at the borders of the growth cartilage. Epiphysiodesis would then be caused by different rates of regeneration in different tissues. It also seems that the smaller the growth cartilage is in size the better is it regenerated after vascular injury such as devascularization or transplantation and the smaller is the risk of a central epiphysiodesis. Consequently if epiphysial transplantation is used in clinical orthopaedic surgery it should be performed as early as possible.

The series of rabbits subjected to lumbar sympathectomy after devascularization is too small to permit any very reliable conclusions but it seems that sympathectomy cannot compensate even partially for the retardation of growth caused by devascularization.

In the study on the effect of temporary ischaemia on longitudinal growth it was found that 3 hours of ischaemia had already caused widening of the epiphysial lines 24 hours after the release of the constricting band. Histologically swelling of cells and disarrangement of the cell columns was seen. Four, 5, 6 and 7 hours of ischaemia produced even more pronounced changes observed 24 hours after the procedure both roentgenologically and histologically. However 5 hours of ischaemia had caused no retardation of growth of the tibia 6 weeks after the procedure and no pathological changes in the growth cartilages were observed at histological examination. Six hours of ischaemia led to some retardation of growth and 7 hours of ischaemia had caused even more retardation 6 weeks after the period of ischaemia however no definite pathological changes were seen in the growth cartilages of these limbs.

the other tissues of which (skin nerves muscles) were seen to be severely damaged at macroscopic examination

Postmortal angiograms showed impaired filling of peripheral vessels after 2 hours of ischaemia the changes after this period of ischaemia were slight but with increasing length of the period of ischaemia it became more and more apparent that changes in the peripheral vessels persisted for up to 14 days. Consequently the retardation of growth found after 6 and 7 hours of ischaemia cannot be attributed solely to the period of ischaemia since impaired circulation for some time after this period probably played a part in producing the injury to the growth cartilages. It was not considered necessary to pursue these experiments further it had been established that 7 hours of ischaemia were needed to cause definite retardation of growth seen 6 weeks after the procedure it had also been established that such a period of ischaemia caused impaired filling of peripheral vessels which probably contributed to the retardation of growth.

It seems that even if the growth cartilage cells are as sensitive to ischaemia as other tissues they possess a remarkable power of regeneration which makes it possible for them to stand vascular injuries with a surprisingly small loss of growth potential whereas other tissues in the limbs are definitely and irreparably injured under similar circumstances.

## CONCLUSIONS

Conclusions based on the results of my experiments on rabbits are as follows

motor and combined motor and sensory denervation by spinal nerve root section leads at most to slight retardation of growth in the hind limb

sensory denervation by means of section of posterior nerve roots leads to retardation of growth of the femur through traumatization of an anaesthetic limb where the motor supply is intact

subtotal devascularization by severance of the large vessels and the muscles in the thigh causes severe interference with longitudinal growth through necrosis of the growth cartilages and particularly in the upper end of the tibia it causes the establishment of bony epiphysiodesis this retardation of growth does not seem to be affected by lumbar sympathectomy

temporary ischaemia must be maintained for 6—7 hours to cause significant retardation of growth though the cells of the growth cartilages are injured they apparently possess a high power of regeneration

The mechanism by which retardation of growth occurs in limbs paralyzed by poliomyelitis still remains obscure this study suggests that motor paralysis (with the sympathetic nerve supply intact) plays a very minor part in the retardation of growth From clinical evidence it seems probable that chronic vascular insufficiency with consequent hypoxia of growth cartilage cells is an important factor in poliomyelitic retardation of growth the possibility of a direct affection of the blood vessels should be kept in mind

## SUMMARY

Paralytic poliomyelitis has been a common cause of inequality of length in the lower limbs. There is a correlation between the degree of paresis and the retardation of growth though considerable deviations are observed and the role of the vascular disturbances in causing retardation of growth in poliomyelitic limbs has been emphasized. However the pathogenesis of the disturbances in growth occurring after poliomyelitis is still obscure. In many other diseases the influence of nervous and vascular factors on bone growth can be demonstrated. *The purpose of this investigation was to shed further light on the relation between the longitudinal growth and the nervous and vascular supply of bone.*

The effects of the following procedures on longitudinal growth of the femur and tibia in the rabbit were investigated

- 1) motor denervation
- 2) motor and sensory denervation
- 3) sensory denervation
- 4) sensory denervation and protection of the denervated limb by insertion under the abdominal skin
- 5) subtotal devascularization by severance of the muscles and vessels in the thigh
- 6) severance of all muscles in the thigh
- 7) severance of the femoral vessels
- 8) subtotal devascularization and lumbar sympathectomy
- 9) temporary ischaemia

The experiments were carried out on rabbits 13-17 days old. Denervation was performed by severing nerve roots of the left hind limb devascularization by severing all muscles and all vessels except those of the skin in the left thigh. Sympathectomy was performed by the trans abdominal route. Temporary ischaemia was maintained by tying a piece of rubber tubing round the left thigh. Useful data were obtained from 132 animals.

*Motor denervation by means of section of 3 or 4 lumbosacral motor*

*nerve roots caused almost complete paralysis of one hind limb and severe atrophy of muscles but only slight retardation of growth of the femur (5 per cent or less) and almost none of the tibia (Fig 3, page 19 Fig 4 page 19)* Section of 2 lumbosacral motor nerve roots caused definite atrophy of muscles and osteoporosis in the affected limb but no significant retardation of growth was found (Fig 5, page 21) The results of section of 4 motor and 4 sensory nerve roots were similar to the results of section of 4 motor nerve roots

Section of 4 sensory nerve roots had caused deformation and retardation of growth of the femur 25 days after operation (Fig 7 page 23) the roentgenological examination of the bones suggested the effect of trauma (Fig 13 page 27) and histological examination revealed a metaphysial fracture (Fig 17 page 29) in some instances the fracture probably involved the growth cartilage As it was assumed that the fracture was the result of inadvertent traumatization of an anaesthetic limb the denervated limb was protected in one series of animals by being inserted under the abdominal skin No fractures and at most slight retardation of growth (Fig 8 page 23) were seen in these animals

*Subtotal devascularization by severance of muscles and vessels in the thigh caused retardation of growth 10-15 per cent in the femur and more in the tibia (Fig 18 page 32)* Histological examination revealed necrosis of the cells of the growth cartilages 24 hours after operation (Fig 32 page 47) 48 hours later considerable regeneration was found at the periphery of the growth cartilage (Fig 34 page 49) regeneration then proceeded towards the centre of the cartilage plate (Fig 35 page 50) Rapid revascularization was seen in postmortal angiograms (Fig 27 page 41 Fig 28 page 41) On the 7th day after devascularization a forked growth cartilage was seen (Fig 36 page 51) this was due to necrotic cartilage cells being left behind in the metaphysis

In some devascularized bones histological examination revealed a split in the growth cartilage in the zone of hypertrophied cells (Fig 33 page 48) It was thought that the split represented a pathological weakness in a growth cartilage deprived of its blood supply consequently this finding might have some bearing upon the pathological processes in slipped upper femoral epiphysis

In animals surviving subtotal devascularization for more than 21 days the upper articular surface of the tibia was usually distorted and cup shaped in these distorted bones a broad central bony bridge joined the epiphysis and the metaphysis (Fig 40 page 54) In some instances the upper end of the tibia was not distorted histological examination revealed

narrow bony bridges and these did not retard longitudinal growth so severely (Fig 39 page 53)

Lumbar sympathectomy did not compensate for the retardation of growth in devascularized limbs

*Temporary ischaemia did not significantly affect longitudinal growth if maintained for up to 5 hours 6 hours of ischaemia produced some retardation of growth and 7 hours even more (Fig 22 page 37) Postmortal angiograms revealed vascular changes after the period of ischaemia (Fig 31 page 44) It was realized that the retardation of longitudinal growth might be due not only to the period of ischaemia but also to a more lasting impairment of blood supply caused by vascular injury*

## REFERENCES

- Abramson D I Flachs K Friesberg J & Mirsky I A* Blood flow in extremities affected by anterior poliomyelitis *Arch intern Med* 71 391-396 1943
- Allison V & Brooks B* Bone atrophy in experimental and clinical study of the changes in bone which result from non use *Surg Gynec Obstet* 33 250-260 1921
- Arkin A M & Katz J F* The effects of pressure on epiphyseal growth The mechanism of plasticity of growing bone *J Bone Jt Surg* 38 A 1056-1076 1956
- Armstrong W D* Bone growth in paralyzed limbs *Proc Soc exp Biol (N Y)* 61 358-362 1946
- Aron M & Simon R* Recherches experimentales sur les facteurs d'accroissement des os longs *Arch Anat (Strasbourg)* 2 207-253 1923
- Axhausen G* Ueber den histologischen Vorgang bei der Transplantation von Gelenkenden insbesondere uiber die Transplantationsfähigkeit von Gelenkknorpel und Epiphysknorpel *Arch klin Chir* 99 1-50 1912
- Bacq Z M* The action of abdominal sympathectomy on the growth of the albino rat and the weight of the genital organs *Amer J Physiol* 95 601-604 1930
- Banks S H & Compere E L* Regeneration of epiphyseal cartilage *Ann Surg* 114 1076-1084 1941
- Barr J S* Growth and inequality of leg length in poliomyelitis *New Engl J Med* 238 737-743 1948
- Barr J S Stinchfield A J & Resdy J A* Sympathetic ganglionectomy and limb length in poliomyelitis *J Bone Jt Surg* 32 A 793-802 1950
- Bergmann E* Ueber das Längenwachstum der Knochen *Dtsch Z Chir* 233 149-194 1931
- Bermann E J* Die Syringomyelie im Kindesalter *Mtschr Kinderheilk* 37 1-14 1927
- Bisgard J D* Effect of sympathetic ganglionectomy upon bone growth *Proc Soc exp Biol (N Y)* 49 229-230 1941
- Bisgard J D* Longitudinal bone growth The influence of sympathetic denervation *Ann Surg* 97 374-380 1933
- Bisgard J D & Bisgard M E* Longitudinal growth of long bones *Arch Surg (Chicago)* 31 568-578 1935
- Boerema I* Ueber das Knochenwachstum *Acta neerl Morph* 4 365-377 1941/42
- Bohlman H R* Experiments with foreign materials in the region of the epiphyseal cartilage plate of growing bones to increase their longitudinal growth *J Bone Jt Surg* 11 365-384 1929
- Bombelli R* Effetti della simpaticectomia lombare sulle modificazioni del circolo in arti inferiori poliomeletici *Arch Ortop (Milano)* 70 73-281 1957



- Borel G. Leber abnormes Laengenwachstum der Knochen (Elongation) infolge venoeser Stauung Inauguraldissertation Zurich 1922
- Braibanti T. Die Arteriographie der Extremitäten bei den Folgen der Heine Medinschen Krankheit Fortschr Rontgenstr 89 77-280 1958
- Brashear H R Jr Epiphyseal fractures A microscopic study of the healing process in rats J Bone Jt Surg 41 A 1055-1064 1959
- Brodin H. Longitudinal bone growth. The nutrition of the epiphyseal cartilages and the local blood supply Acta orthop scand suppl 20 1955
- Brookes M. Femoral growth after occlusion of the principal nutrient canal in day-old rabbits J Bone Jt Surg 39-B 563-571 1957
- Brookes M & Harrison R G. The vascularization of the rabbit femur and tibiofibula J Anat (Lond) 91 61-72 1957
- Brücke F. Zur Frage der Bedeutung des Epiphysenfugenknorpels für das Wachstum der langen Röhrenknochen Virchows Arch path Anat 279 641-650 1931
- Bunak I I & Klebanov E A. Effect of increased mechanical load upon formation of extremital bones in growing animals Ark Anat Gistol Embriol 38 (5) 43-50 1960
- Bunnell S. Surgery of the hand J B Lippincott Company Philadelphia 1944
- Campbell C J. Crisolia A & Zanconato G. The effects produced in the cartilaginous epiphyseal plate of immature dogs by experimental surgical traumata J Bone Jt Surg 41 A 121-1240 1959
- Cannon W B, Newton H F, Bright E M, Menkin V & Moore R M. Some aspects of the physiology of animals surviving complete exclusion of sympathetic nerve impulses Amer J Physiol 89 84-107 1919
- Carpenter E B & Dalton J B. A critical evaluation of a method of epiphyseal stimulation J Bone Jt Surg 38 A 1089-1097 1956
- Chapchal C & Zeldenrust J. Experimental research for promoting longitudinal growth of the lower extremities by irritation of the growth region of femur and tibia Acta orthop scand 27 311-396 1948
- Collins V J, Foster W L & West H J. Vasomotor disturbances in polyomyelitis with special reference to treatment with paravertebral sympathetic block New Engl J Med 236 644-697 1947
- Combes M A, Clark W A, Gregory C F & James J A. Sciatic nerve injury in infants J Amer med Ass 173 1336-1339 1960
- Compere E L & Adams C O. Studies of longitudinal growth of long bones J Bone Jt Surg 19 922-936 1937
- Cooley J C, Mussey R D & Rogers J C T. Femoral arteriovenous fistula creation in the treatment of the short leg Experience with seven cases Arch Surg (Chicago) 80 838-841 1960
- Corbin A B. Alterations in the hip joint after deafferentation Arch Surg (Chicago) 35 1145-1158 1937
- Corbin A B & Hinsey J C. Influence of the nervous system on bone and joints Anat Rec 75 307-317 1939
- Curcio Cited by R Cassirer. Die vasomotorisch trophischen Venosen S Karger Berlin 1901
- De Castro F. Quelques observations sur l'intervention du système nerveux autonome dans l'ossification Innervation du tissu osseux et de la moelle osseuse Trav Lab Rech biol Univ Madr 26 215-244 1930

- De Forest R E Herrick J F Janes J M & Krusen F H* Effects of ultrasound on growing bone. An experimental study. *Arch phys Med* 34 21-31 1953
- Dickinson P H* Venous stasis and bone growth. *Lap Med Surg* 11 49-53 1953
- Doerr G M & Janes J M* Effect of arteriovenous fistula and ligation of proximal vein on the growth of bone. *Proc Mayo Clin* 34 555-560 1959
- Dohn A* Functional plethysmography in disturbances of the circulation following poliomyelitis. *Acta orthop scand* 22 337-338 1953
- Dubreuil G* La croissance des os des mammifères. II Croissance au niveau du cartilage de conjugaison. *C R Soc Biol (Paris)* 74 888-890 1913
- DuHamel H L* Sur le développement et la crue des os des animaux. *Mémoires de mathématique et de physique tirés des Registres de l'Académie Royale des Sciences* 55 354-370 1747
- Duthie R B* The significance of growth in orthopaedic surgery. *Clin Orthop* 14 7-19 1959
- Eggers G W V Shindler T O & Pomeroy C M* The influence of the contact compression factor on osteogenesis in surgical fractures. *J Bone Jt Surg* 31 A 693-716 1949
- Elo J* The effect of subperiosteally implanted autogenous whole thickness skin graft on growing bone. *Acta orthop scand suppl* 45 1960
- Eloesser L* On the nature of neuropathic affections of the joints. *Ann Surg* 66 201-207 1917
- Enderlen* Zur Reimplantation des resezierten Intermediärknorpels beim Kaninchen. *Dtsch / Chir* 51 574-598 1899
- Eians F G* Stress and strain in bones. Charles C Thomas Springfield Ill 1957
- Fahey J J* The effect of lumbar sympathetic ganglionectomy on longitudinal bone growth as determined by the telorontgenographic method. *J Bone Jt Surg* 18 1042-1046 1935
- Fancom G* Peripheral nutritional disorders. *J Bone Jt Surg* 41 B 885-886 1959
- Fell H B* Skeletal development in tissue culture. In *The biochemistry and physiology of bone*. Edited by G H Bourne. Academic Press Inc New York 1956
- Fell H B & Robison R* The growth development and phosphatase activity of embryonic avian femora and limb buds cultivated in vitro. *Biochem J* 3 767-784 1959
- Ferguson A B* Surgical stimulation of bone growth by a new procedure. *J Amer med Ass* 100 46-47 1933
- Ferguson A B Jr & Akahoshi Y* Vascular patterns in immobilized denervated or devascularized rabbit limbs. *J Bone Jt Surg* 42 A 617-624 1960
- Fischer* Ueber trophische Störungen nach Nervenverletzungen an den Extremitäten. *Berl klin Wschr* 8 145-147 1871
- Ford F R* Diseases of the nervous system in infancy childhood and adolescence. 4th ed. Blackwell Scientific Publications Oxford 1960
- Ford L T & Canales G M* A study of experimental trauma and attempts to stimulate growth of the lower femoral epiphysis in rabbits - III. *J Bone Jt Surg* 42 A 439-446 1960

- Ford L T & Key J I A study of experimental trauma to the distal femoral epiphysis in rabbits J Bone Jt Surg 38 A 84-9 1956
- Friedl E & Schin H R Zur Frage der Knochenatrophie Ergebn med Strahlenforsch 1 95-130 1925
- Gatewood & Mullen B P Experimental observations on the growth of long bones Arch Surg (Chicago) 15 215-221 1927
- Geiser V Muskelaktion und Tätigkeit der Knochenwachstumszone Z Orthop 89 194-205 1937
- Gelbke H The influence of pressure and tension on growing bone in experiments with animals J Bone Jt Surg 33 A 947-954 1951
- Ghillini C Untersuchungen über den Einfluss der Nervenverletzung auf das Knochenwachstum Z orthop Chir 5 274-7 1898
- Gillespie J I The nature of the bone changes associated with nerve injuries and disuse J Bone Jt Surg 36 B 464-473 1954
- Goet I H DuToit J C & Stuart B H Vascular changes in poliomyelitis and the effect of sympathectomy on bone growth Acta med scand suppl 306 56-83 1955
- Gretelle V R & Jones J W An experimental study of overgrowth after fracture Surg Gynec Obstet 105 717-721 1957
- Grey E G & Carr C L An experimental study of the factors responsible for non infectious bone atrophy Pull Johns Hopk Hosp 26 381-385 1915
- Gullickson G Jr Olson W & Holtke F J The effect of paralysis of one lower extremity on bone growth Arch phys Med 31 39-400 1950
- Gullickson G Jr Kubicek W G & Holtke F J Effects of stimulation of lumbar sympathetic nerves on longitudinal bone growth in dogs Fed Proc 10 56 1951
- Haas S L The transplantation of the articular end of bone including the epiphyseal cartilage line Surg Gynec Obstet 53 301-332 1916
- Haas S L The changes produced in the growing bone after injury to the epiphyseal cartilage plate J orthop Surg 1 67-99 166-179 226-239 1917
- Haas S L The relation of the blood supply to the longitudinal growth of bone Amer J orthop Surg 15 157-171 305-316 1917
- Haas S L Stimulation of bone growth Amer J Surg 95 125-131 1958
- Hagelstam L Paresis of the urinary bladder in poliomyelitis Ann Chir Gynaec Penn 45 103 1956
- Hales S Statick der Gewächse Rengerische Buchhandlung Halle 1,48
- Haller I First lines of physiology Charles Elliot Edinburgh 1786
- Harris H I quoted by Lindsay D T Control of human epiphyseal growth History of studies of growth of bone Univ Minn med Bull 20 450-469 1949
- Harris R I The effect of lumbar sympathectomy on the growth of legs shortened from anterior poliomyelitis J Bone Jt Surg 12 859-866 1930
- Harris R I & McDonald J L The effect of lumbar sympathectomy upon the growth of legs paralyzed by anterior poliomyelitis J Bone Jt Surg 18 35-45 1936
- Harris W R & Hobson A W Histological changes in experimentally displaced upper femoral epiphyses in rabbits J Bone Jt Surg 35 B 914-921 1950
- Heikel H I I Aplasia and hypoplasia of the radius Studies on 64 cases and

- De Forest R E Herrick J F Janes J M & Krusen F H* Effects of ultra sound on growing bone. An experimental study. *Arch phys Med* 34 21-31 1953
- Dickinson P H* Venous stasis and bone growth. *Lxp Med Surg* 11 49-53 1953
- Doerr G M & Janes J M* Effect of arteriovenous fistula and ligation of proximal vein on the growth of bone. *Proc Mayo Clin* 34 555-560 1959
- Dohn A* Functional plethysmography in disturbances of the circulation following poliomyelitis. *Acta orthop scand* 2 337-338 1953
- Dubreuil G* La croissance des os des mammifères. II Croissance au niveau du cartilage de conjugaison. *C R Soc Biol (Paris)* 74 888-890 1913
- DuHamel H L* Sur le développement et la crue des os des animaux. *Memoires de mathematique et de physique tirez des Registres de l'Academie Royale des Sciences* 55 354-370 1747
- Duthie R B* The significance of growth in orthopaedic surgery. *Clin Orthop* 14 7-19 1959
- Eggers G H V Shindler T O & Pomeroy C M* The influence of the contact compression factor on osteogenesis in surgical fractures. *J Bone Jt Surg* 31 A 693-710 1949
- Elo J* The effect of subperiosteally implanted autogenous whole thickness skin graft on growing bone. *Acta orthop scand suppl* 45 1960
- Eloesser L* On the nature of neuropathic affections of the joints. *Ann Surg* 66 201-207 1917
- Enderlen* Zur Reimplantation des resezierten Intermediärknorpels beim Kaninchen. *Dtsch J Chr* 51 574-598 1899
- Evans F G* Stress and strain in bones. Charles C Thomas Springfield Ill 1957
- Fahey J J* The effect of lumbar sympathetic ganglionectomy on longitudinal bone growth as determined by the teleoroentgenographic method. *J Bone Jt Surg* 18 104-1046 1935
- Fanconi G* Peripheral nutritional disorders. *J Bone Jt Surg* 41 B 885-886 1959
- Fell H B* Skeletal development in tissue culture. In *The biochemistry and physiology of bone*. Edited by G H Bourne. Academic Press Inc New York 1956
- Fell H B & Robison R* The growth development and phosphatase activity of embryonic avian femora and limb buds cultivated in vitro. *Biochem J* 23 767-784 1929
- Ferguson A B* Surgical stimulation of bone growth by a new procedure. *J Amer med Ass* 100 26-27 1933
- Ferguson A B Jr & Akahoshi Y* Vascular patterns in immobilized denervated or devascularized rabbit limbs. *J Bone Jt Surg* 41 A 617-624 1960
- Fischer* Leber trophische Störungen nach Nervenverletzungen an den Extremitäten. *Berl klin Wschr* 8 145-147 1871
- Ford F R* Diseases of the nervous system in infancy childhood and adolescence. 4th ed Blackwell Scientific Publications Oxford 1960
- Ford L T & Canales G M* A study of experimental trauma and attempts to stimulate growth of the lower femoral epiphysis in rabbits. - III. *J Bone Jt Surg* 41 A 439-446 1960

- Janes J M & Musgrove J E* Effect of arteriovenous fistula on growth of bone  
Surg Clin N Amer 30 1191—1200 1930
- Jansen M* quoted by *Lindsay D T* Control of human epiphyseal growth  
History of studies of growth of bone Univ Minn med Bull 0 450—469 1949
- Joseph H* Ueber den Einfluss der Nerven auf Ernährung und Neubildung Arch  
Anat Physiol Wiss Med 1872 206—236
- Kapsammer G* Das Verhalten der Knochen nach Ischiadicusdurchschneidung  
Arch Klin Chir 36 348—360 1898
- Kassowitz M* Die Bildung und Resorption des Knochengewebes und das Wesen  
der rachitischen Knochenerweichung Zbl med Wiss 16 785—91 18,8
- Kelly P J* *Janes J M & Peterson L F* 4 The effect of arteriovenous fistulae  
on the vascular pattern of the femora of immature dogs J Bone Jt Surg 41 A  
1101—1108 1959
- Kemp C R Tuttle W W & Hines H M* Studies on the temperature character-  
istics blood flow and activity in normal and denervated limbs of the dog Amer  
J Physiol 150 705—711 1947
- Kishikawa E* Studien über einige lokale Reize welche das Längenwachstum des  
Langrohrenknochens steigern Fukuoka Acta med 29 4 1936
- Kornew P G* Transplantation und Knochenwachstum Experimentelle Unter-  
suchung Arch Klin Chir 154 499—564 1929
- Kottke F J Gullickson G & Olson M E* Studies on the disturbance of longi-  
tudinal bone growth II Effect of the sympathetic nervous system on longitu-  
dinal bone growth after acute anterior poliomyelitis Arch phys Med 39  
770—779 1958
- Krause W* Die Anatomie des Kaninchens in topographischer und operativer  
Rucksicht Wilhelm Engelmann Leipzig 1884
- Krepler P* Angeborene einseitige spinale Vasomotorenlähmung mit trophischen  
Störungen Z Kinderheilk 81 171—180 1958
- Kritter A E & Blount W P* A study of the growth of human epiphyses of the  
tibia and femur Surg Forum 10 808—810 1960
- Kuré A* Die Frage nach der trophischen Innervation des passiven Gewebes durch  
Spinalparasymphicus Klin Wschr 15 822—825 1936
- Lacroix P* The organization of bones J & A Churchill Ltd London 1951
- Launbeck B* Ueber krankhaftes Längenwachstum der Rohrenknochen und  
seine Verwerthung für die chirurgische Praxis Berl Klin Wschr 6 265—270  
1869
- Langenskiöld A* Tibia vara (osteocondrosis deformans tibiae) Acta chir scand  
103 1—22 1952
- Langenskiöld A* Inhibition and stimulation of growth Acta orthop scand 26  
308—316 1957
- Lassek A M & Moyer E A* An ontogenetic study of motor deficits following  
dorsal brachial rhizotomy J Neurophysiol 16 247—251 1953
- Latarjet A* Resultats experimentaux sur l'accroissement des os en longueur  
CR Ass Anat 14 72—90 1912
- Lerique J* Modifications de la croissance osseuse au cours de la poliomyélite  
La croissance osseuse dans les premiers mois J Radiol Electrol 37 110—111  
1950
- Letander G* Ueber die Behandlung von Brüchen des Oberschenkelknochens nebst

- Beitrag zur Kenntnis des gesteigerten Längenwachstums der Rohrknochen der unteren Extremitäten nach Bruch derselben Acta chir scand suppl 12 1929
- Liek L Ueber den Einfluss der arteriellen Hyperaemie auf die Regeneration Arch klin Chir 67 2 9-259 1902
- Lindholm R On inequality in length between lower limbs after poliomyelitis with unilateral involvement acquired during the growth period Acta orthop scand 1951 (in press)
- Louenstein J M Pauporte J Richards I & Davison R Effects of sympathectomy on blood turnover rates in muscle and bone Surgery 43 768-773 1958
- Lundback A Experimental investigations on the function of the autonomic nervous system during the acute phase of poliomyelitis Acta med Scand 114 565-583 1943
- Maass H Zur Frage über das Knochenwachstum Arch klin Chir 14 198-207 1872
- Mason M L & Bell J L The treatment of open injuries to the hand Surg Clin N Amer 36 1337-1361 1956
- Watson D D Radical treatment of craniopharyngioma Paper read at the 15th annual meeting of the Scandinavian Neurosurgical Society Lund Sweden September 9-10 1960
- McCullagh G P McGadden G D & Milroy T H The effects produced by unilateral lumbar sympathectomy I Cutaneous temperatures and the limb musculature J Physiol (Lond) 69 353-363 1930
- McPherson I & Kessel A H L Asymmetry of blood flow in the feet in anterior poliomyelitis Clin Sci 14 361-380 1955
- Meisenbach R O A consideration of the chemical and mechanical stimulation of bone with reference to the epiphyseal and diaphyseal lines Results of animal experimentation Amer J orthop Surg 8 78-94 1910
- Miescher F De inflammatione ossium eorumque anatomie generali G Fichler Berolino 1836
- Milne Edwards A Etudes chimiques et physiologiques sur les os Ann Sci nat Zool 13 113-192 1860
- Moldaver J Analysis of neuromuscular disorders in poliomyelitis J Bone Jt Surg 46 103-115 1944
- Montgomery W S & Ingram A J Experimental studies and clinical evaluation of linear growth stimulation Sth med J (Bgham Ala) 49 793-797 1956
- Morgan J D Blood supply of growing rabbit's tibia J Bone Jt Surg 41 B 185-203 1959
- Müller H Ueber die Entwicklung der Knochensubstanz nebst Bemerkungen über den Bau rachitischer Knochen Z wiss Zool 9 147-233 1858
- Müller H Die normale und pathologische Physiologie des Knochens Johann Ambrosius Barth Leipzig 1944
- Nasse H Ueber den Einfluss der Nervendurchschneidung auf die Ernährung insbesondere auf die Form und die Zusammensetzung der Knochen Pflügers Arch ges Physiol 23 361-405 1880
- Note Josseland G Des troubles de l'accroissement des os consecutifs aux lesions des cartilages de conjugaison Rev Chir (Paris) 14 385-403 1894

- Ogilvie W H The place of operations upon the sympathetic system in the treatment of poliomyelitis *Proc roy Soc Med* 26 429-435 1933
- Ollier L Traite experimental et clinique de la regeneration des os et de la production artificielle du tissu osseux Vol I Victor Masson & Fils Paris 1867
- Paletta F A Hillman V & Ship A G Prolonged tourniquet ischemia of extremities An experimental study on dogs *J Bone Jt Surg* 4 A 945-949 1960
- Pauporte J Lowenstein J M Richards I & Davison R Blood turnover rates distal to an arteriovenous fistula *Surgery* 43 828-836 1958
- Pearse H E Jr & Morton J J The stimulation of bone growth by venous stasis *J Bone Jt Surg* 12 97-111 1930
- Pearse H E Jr & Morton J J The influence of alterations in the circulation on the repair of bone *J Bone Jt Surg* 13 68-74 1931
- Pease C A Local stimulation of growth of long bones *J Bone Jt Surg* 34 A 1-23 1952
- Pemister D B Bone growth and repair *Ann Surg* 102 261-285 1935
- Paulachs P & Vidal Barraquer F Ulceres poliomyelitiques *Lyon chir* 51 679-69- 1956
- Policard A A propos des mecanismes de la croissance osseuse *Presse med* 1 345-348 1930
- Pottorf J L An experimental study of bone growth in the dog *Anat Rec* 10 234-235 1916
- Potts W J The pathology of Charcot joints *Ann Surg* 86 596-606 19 7
- Pratt C W M & McCance R A Severe undernutrition in growing and adult animals 2 Changes in the long bones of growing cockerels held at fixed weights by undernutrition *Brit J Nutr* 14 75-84 1960
- Prick J J G On morbidly changed blood vessels as the central aspect of the pathologic anatomy of poliomyelitis also in connection with clinical observations *Volia psychiat (Amst)* 61 593-604 1958
- Pye Smith P H Observations upon the persistent effects of division of the cervical sympathetic *J Physiol (Lond)* 8 25-49 1887
- Pylkkanen P I Coxa vara infantum *Acta orthop scand suppl* 48 1960
- Ratliff A H C The short leg in poliomyelitis *J Bone Jt Surg* 41 B 56-69 1959
- Rehn E & Wakabayashi Die homoplastische Transplantation des Intermediar Knorpels im Thierexperiment *Arch klin Chir* 97 1-34 1912
- Reichert F L The importance of circulatory balance in the survival of replanted limbs *Bull Johns Hopk Hosp* 49 86-93 1931
- Remak E Neuritis und Polyneuritis In *Specielle Pathologie und Therapie* herausgegeben von H Nothnagel Band VI 3 Alfred Holder Wien 1904
- Richards I & Stoffer R The stimulation of bone growth by internal heating *Surgery* 46 84-96 1959
- Rine P A Shortening and paralysis in poliomyelitis *Lancet* II 980-983 1957
- Rine P A Experimental bone lengthening by epiphyseal distraction *Brit J Surg* 46 169-173 1958
- Rine P A Prognosis of limb inequality following paralytic poliomyelitis *Lancet* II 1306-1308 1958
- Rine P A The influence of the nervous system upon the growth of bones *J Bone Jt Surg* 43 B 121-140 1961

- Ring P A & Lee J The effect of heat upon the growth of bone J Path Bact 75 405-412 1958
- Ring P A & Ward B C H Paralytic bone lengthening following poliomyelitis Lancet II 551-553 1958
- Robertson D E Sympathectomy in children Surg Gynec Obstet 58 312-317 1934
- Sabin A B Pathology and pathogenesis of human poliomyelitis J Amer med Ass 120 506-511 1942
- Sabin A B & Ward R The natural history of human poliomyelitis I Distribution of virus in nervous and non nervous tissues J exp Med 73 771-793 1941
- Samuel S Das Gewebswachsthum bei Storungen der Innervation Virchows Arch path Anat 113 272-314 1888
- Schiff Recherches sur l'influence des nerfs sur la nutrition des os C R Acad Sci (Paris) 38 1050-1055 1854
- Schroder H & Seyfarth H Untersuchungen uber die Durchblutung im Gipsverband Z Orthop 93 283-288 1960
- Schubert A Wachstumsunterschiede und atrophische Vorgänge am Skelettsystem Dtsch Z Chir 161 80-123 1911
- Seeligmuller A Ueber Elongation der Knochen an gelähmten Extremitäten Zbl Chir 6 465-468 1879
- Selby D & Murray P D F Grafts of longitudinal halves of limb buds of the four day chick Aust J exp Biol med Sci 5 181-186 19 8
- Selye H On the mechanism controlling the growth in length of the long bones J Anat (Lond) 68 289-292 1934
- Selye H & Bajusz E Effect of denervation on experimentally induced changes in the growth of bone and muscle Amer J Physiol 19 297-300 1938
- Serelle Stase veineuse et croissance osseuse Bull Acad nat Med (Paris) 132 471-474 1948
- Silfverskiöld V Über Längenwachstum der Knochen und Transplantation von Epiphysenscheiben Experimentelle Arbeit Acta chir scand 75 77-104 1934
- Simon R Innervation sympathique et croissance C R Soc Biol (Paris) 113 I 709-710 1930
- Sissons H A The growth of bone In The biochemistry and physiology of bone Edited by G H Bourne Academic Press Inc New York 1956
- Smith E Rosenblatt P & Limauro A B The role of the sympathetic nervous system in acute poliomyelitis J Pediat 34 1-11 1949
- Smith W S & Cunningham J B The effect of alternating distracting forces on the epiphyseal plates of calves Clin Orthop 10 125-130 1957
- Sousa Pereira L influence des troubles de la vascularisation sur la croissance des os en longueur Etude expérimentale Rev Orthop 25 140-154 1938
- Spencer W A Jackson R R Vallbona C & Harrison G M Circulatory disturbances in life threatening poliomyelitis Amer Heart J 59 384-400 1960
- Steindler A The newer pathological and physiological concepts of anterior poliomyelitis and their clinical interpretation J Bone Jt Surg 9 59-76 1947
- Stenfort K Treatment of cold feet and legs after poliomyelitis Angiology 2 345-349 1951
- Stinchfield A J Reidy J A & Barr J S Prediction of unequal growth of the



- lower extremities in anterior poliomyelitis J Bone Jt Surg 31 A 48-484 1949
- Stirling W* Note on the effects of division of the sympathetic nerve of the neck in young animals J Anat Physiol 20 311-312 1876
- Strobino I J French G O & Colonna P C* The effect of increasing tensions on the growth of epiphyseal bone Surg Gynec Obstet 95 694-700 1952
- Sulzer E* Ueber den Einfluss der sensiblen Innervation auf den ausgewachsenen Knochen Dtsch Z Chir 193 186-193 1925
- Suzuki H K* Synergistic effect of thyroxin and estradiol valerate on the inhibition of linear growth in the mouse femur Growth 23 291-302 1959
- Telford E D & Stopford J S B* Some experiences of sympathectomy in anterior poliomyelitis Brit med J II 770-772 1933
- Thoma R* Synostosis suturae sagittalis cranii Ein Beitrag zur Histomechanik des Skeletts und zur Lehre von dem interstitiellen Knochenwachstum Virchows Arch. path Anat 188 248-360 1907
- Tower S S* Trophic control of non nervous tissue by the nervous system a study of muscle and bone innervated from an isolated and quiescent region of spinal cord J comp Neurol 67 241-267 1937
- Trott A W Hellstrom M D & Green H T* The apparent absence of significant primary vasospasm in acute spinal poliomyelitis Pediatrics 17 230-236 1956
- Trott A W Nestine M D & Green H T* The chronology of circulatory changes in poliomyelitis J Bone Jt Surg 40-A 245-255 1958
- Trueta J* Influence of blood supply in controlling bone growth Bull Hosp Jt Dis (N.Y.) 14 147-157 1953
- Trueta J & Amato V P* The vascular contribution to osteogenesis III Changes in the growth cartilage caused by experimentally induced ischaemia J Bone Jt Surg 4-B 571-587 1960
- Trueta J & Little K* The vascular contribution to osteogenesis II Studies with the electron microscope J Bone Jt Surg 42 B 367-376 1960
- Trueta J & Morgan J D* The vascular contribution to osteogenesis I Studies by the injection method J Bone Jt Surg 42 B 97-109 1960
- Vahlquist B* The longitudinal growth of the long tubular bones in man studied with the aid of lead lines Acta chir scand 88 299-308 1943
- Vauhen J I & Bender L F* Effects of ultrasound on growing bone Arch phys Med 40 158-160 1959
- Volkmann R* Chirurgische Erfahrungen über Knochenverbiegungen und Knochenwachstum Virchows Arch path Anat 44 512-540 1862
- Vulpian A* Leçons sur l'appareil vaso-moteur Tome II Librairie Germer Baillière Paris 1875
- Walker J W Paletta F Y & Cooper T* The relationship of post ischaemic histopathological changes to muscle and subcuticular temperature patterns in the canine extremity Surg Forum 10 836-838 1959
- Wegner C* Ueber das normale und pathologische Wachstum der Rohrknochen Eine kritische Untersuchung auf experimenteller und casuistischer Grundlage Virchows Arch path Anat 61 44-76 1874
- Wile J C & Smithwick P H* The autonomic nervous system Henry Hampton London 1944
- Wiggins H E West F E & O Connell T F* Investigation of disturbances in

- blood flow following acute anterior poliomyelitis *Amer J Surg* 94 155-157 1957
- Wilson C L & Percy E C Experimental studies on epiphyseal stimulation *J Bone Jt Surg* 38 A 1096-1104 1956
- Wilson P D & Thompson T C A clinical consideration of the methods of equalizing leg length *Ann Surg* 110 99-1014 1939
- Winters J C Smith A H & Mendel L B The effects of dietary deficiencies on the growth of certain body systems and organs *Amer J Physiol* 80 576-593 19-7
- Wolff J Ueber die innere Architectur der Knochen und ihre Bedeutung für die Frage vom Knochenwachsthum *Virchows Arch path Anat* 50 389-450 1870
- Wu Y K & Miltner L J A procedure for stimulation of longitudinal growth of bone *J Bone Jt Surg* 19 909-911 1937
- Zimányi I Prohászka M Sondi M & Ormai S Arterial hypertension after poliomyelitis *Acta med scand* 164 497-505 1959





11

12



J R V

I = 10 10 10 10 10

*Wen*

MINERAL METABOLISM  
OF FRACTURES OF THE TIBIA IN MAN  
STUDIED WITH EXTERNAL  
COUNTING OF  $\text{Sr}^{85}$

*by*

**BO WENDEBERG**

MUNKSGAARD

COPENHAGEN 1961





*From the Orthopaedic Research Laboratories (Head Goran C H Bauer  
MD) of the Orthopaedic Clinic Malmö (Head Sophus von Rosen  
MD) Lund University Sweden*

---

MINERAL METABOLISM  
OF FRACTURES OF THE TIBIA IN MAN  
STUDIED WITH EXTERNAL  
COUNTING OF  $\text{Sr}^{85}$

LIBRARY,

Acc No 4098

Cl No

Date of Acc 20 12-62

by

BO WENDEBERG







## Table of Contents

|  |    |
|--|----|
| Introduction   | 9  |
| CHAPTER I. Tracer methods for study of mineral metabolism in fractures | 10 |
| 1. Choice of tracer  | 10 |
| 2. Applications of radioactive tracers                                 | 11 |
| a. Studies in experimental animals                                     | 11 |
| b. Studies in man  | 12 |
| 3. Interpretation of tracer data                                       | 13 |
| 4. Purpose of this investigation                                       | 14 |
| CHAPTER II. Investigation  | 15 |
| A. Clinical material   | 15 |
| B. Method  | 16 |
| 1. Dose and administration of Sr                                       | 16 |
| 2. External counting measurements                                      | 16 |
| 3. Equipment   | 19 |
| 4. Statistics  | 19 |
| 5. General mineral metabolic studies                                   | 20 |
| C. Results   | 20 |
| 1. Intact bone   | 20 |

|   |           |
|---|-----------|
| 2 Normally healing fractures  | 22        |
| a Variation in uptake of $\text{Sr}^{85}$ with age of fracture                    | 22        |
| b Activity ratios fracture/intact leg 14 days after injection of $\text{Sr}^{85}$ | 27        |
| 3 Fractures with delayed or non union   | 31        |
| <b>CHAPTER III Discussion</b>   | <b>35</b> |
| A Kinetics of strontium in man  | 35        |
| B Interpretation of external counting of $\text{Sr}^{85}$                         | 39        |
| C Changes in the accretion rate induced by the fracture                           | 47        |
| 1 Accretion rate in the fracture region   | 47        |
| a Normally healing fractures  | 47        |
| b Fractures with delayed or non union   | 48        |
| c Absolute accretion rate   | 49        |
| 2 Accretion rate in bone adjacent to the fracture                                 | 50        |
| <b>CHAPTER IV Summary and Conclusions</b>   | <b>52</b> |
| Zusammenfassung   | 55        |
| Resume  | 58        |
| Acknowledgements  | 60        |
| References  | 61        |
| Tables  | 65        |
| Radiographs   | 71        |

## INTRODUCTION

The orthopaedic surgeon every day witnesses the remodeling capacity of the skeleton. He knows that this capacity is stronger in childhood than in adults and that it is particularly strong during healing of fractures. He does not know, however, the rates of the two processes of remodeling: bone formation and resorption. The reason for this is simply that he cannot measure them. He cannot trace the origin of the organic matrix or the inorganic bone salt which build up the fracture callus. Hence he cannot measure the rate of bone formation in the callus. With the aid of X-ray techniques it is well known that the metaphyseal ends of a fractured bone shaft lose mineral. It is not known whether this condition is due to a diminished rate of bone formation or an increased rate of bone resorption or if perhaps both possibilities obtain.

Animal experiments have shown that use of radioactive isotope tracer techniques should make it possible to measure the gross rates of the anabolic and catabolic processes involved in bone healing. Such techniques have now been developed to a point where they are safe and convenient for use in man.

Therefore it was decided to study the mineral metabolism of fracture in man with the aid of a radioactive isotope tracer method.

TRACER METHODS FOR STUDY OF  
MINERAL METABOLISM IN  
FRACTURES

## 1 Choice of tracer

Tracer methods have been fundamental for bone physiology since the days of Belcher Duhamel and Hunter around 1750 (see Bick, 1948). Two factors hampered the use of pre isotope tracers: they lacked specificity and were difficult to measure quantitatively. The organic molecule of alizarin in madder which was used for bone studies by the above authors for example does not follow the metabolic pathways of any single component of the bone tissue, and is hard to measure quantitatively even with modern equipment.

Alizarin porphyrins, tetracyclines, and other materials which stain forming bone are valuable tracers because they identify bone formed since the administration of the tracer. Autoradiography following administration of  $\beta$ -emitting isotopes such as  $\text{Ca}^{45}$  or  $\text{P}^{32}$  gives a picture similar to that obtained with alizarin: the autoradiographic image permits higher resolution and is somewhat less difficult to quantitate. Cortical bone is remodeled by interstitial replacement of Haversian systems. Planimetry of newly formed as compared to old Haversian systems permits calculation of the fraction of the bone replaced (formed) per unit time as has been recently shown by Frost, Villanueva and Roth (1960). Such methods are difficult to apply to cancellous bone because of its distorted structure. In the fracture callus this difficulty is particularly evident.

With radioactive isotopes of calcium, sodium, strontium, or any other



component of the bone salt the problem of quantitation is easy the amount of tracer per unit bone tissue can be measured in for example, ashed specimens of the bone In the same way stable strontium or lead may be used as tracers for the bone salt relative to radioactive tracers they are difficult to measure unless given in large doses

In experimental animals it is not a problem that stable elements or  $\beta$ -emitting isotopes can be assayed practically only in biopsy or autopsy specimens of the skeleton In man it is necessary to make serial measurements *in vivo* During recent years  $\gamma$  emitting isotopes of calcium ( $\text{Ca}$ , half-life 4.9 days) and strontium ( $\text{Sr}$  half life 65 days) have become available Their half-lives are sufficiently short to make them safe for use in man It is now a routine procedure to measure  $\gamma$ -emitting isotopes in the body with the aid of scintillation counters located on the body surface

The metabolic fate of strontium injected into the circulation is not identical with that of calcium (Bauer Carlsson and Lindquist, 1961) The main difference is that strontium is cleared from the plasma by urinary and faecal excretion more efficiently than is calcium (Spencer, Laszlo and Brothers 1957) However studies in animals (Bauer Carlsson and Lindquist 1952a) and in man (Dow and Stanbury, 1960) have failed to demonstrate any difference in the rates at which the skeleton clears plasma from the two elements when given in tracer dosage As  $\text{Ca}$  has not been routinely produced until quite recently  $\text{Sr}$  has been the tracer of choice for studies of fracture metabolism in man Owing to its longer half-life it is still preferable for long term studies

Owing to the bulk of the shielding of the scintillation crystal, external counting techniques are more suitable for larger animals and man than for small experimental animals

## 2 Applications of radioactive tracers

a *Studies in experimental animals* — The mineral metabolism of fractures has been studied in animals with the aid of radioactive isotopes of calcium phosphorus strontium and other components of the bone salt These studies have shown that the uptake of tracer from the circulation is more rapid in a fracture than in comparable normal bone Bauer (1954) found that the amount of tracer in a one week old fracture of the femoral shaft in adult rats was higher than normal within hours after parenteral injection of  $\text{Ca}$  and increased

TRACER METHODS FOR STUDY OF  
MINERAL METABOLISM IN  
FRACTURES

## 1 Choice of tracer

Tracer methods have been fundamental for bone physiology since the days of Belchier Duhamel, and Hunter around 1750 (see Bick, 1948). Two factors hampered the use of pre-isotope tracers: they lacked specificity and were difficult to measure quantitatively. The organic molecule of alizarin in madder, which was used for bone studies by the above authors, for example, does not follow the metabolic pathways of any single component of the bone tissue and is hard to measure quantitatively even with modern equipment.

Alizarin, porphyrins, tetracyclines, and other materials which stain forming bone are valuable tracers because they identify bone formed since the administration of the tracer. Autoradiography following administration of  $\beta$ -emitting isotopes such as  $\text{Ca}^{45}$  or  $\text{P}^{32}$  gives a picture similar to that obtained with alizarin; the autoradiographic image permits higher resolution and is somewhat less difficult to quantitate. Cortical bone is remodeled by interstitial replacement of Haversian systems. Planimetry of newly formed as compared to old Haversian systems permits calculation of the fraction of the bone replaced (formed) per unit time, as has been recently shown by Frost, Villanueva and Roth (1960). Such methods are difficult to apply to cancellous bone because of its distorted structure. In the fracture callus this difficulty is particularly evident.

With radioactive isotopes of calcium, sodium, strontium, or any other

### 3 Interpretation of tracer data

Radioactive isotopes of calcium and strontium present in the body fluids are incorporated into the skeleton by irreversible deposition (accretion) as a part of formation of bone mineral (Bauer Carlsson and Lindquist 1955 b, 1961). Under certain conditions a correlation was found in experiments in rats, between the amount of activity recovered from different portions of bone and the amount of bone salt formed in these bones in the course of bone growth and repair (Bauer 1961). An increased uptake of labelled calcium or strontium in fractured as compared to intact, bone may thus indicate an increased accretion rate in fractured bone. In man it has been shown how the externally recorded activities following intravenous injection of  $\text{Ca}^{45}$  or  $\text{Sr}^{90}$  may be interpreted as an index of the accretion rate (Bauer and Wendeberg 1969).

On the basis of the general consideration above data from tracer studies in animals may be interpreted as follows:

Shortly after fracture the accretion rate in a fractured bone shaft is increased. It reaches a peak value within week in rats and stays higher than normal for several months. The calcium of the callus is derived from the general circulation. The accretion rate in poorly uniting fractures does not differ from that in normally healing fractures. The mineral loss in adjacent bone induced by a fracture is not caused by a decrease in the accretion rate but by a rise in the rate of bone salt resorption (Bauer and Carlsson 1965). Some data indicate that the loss of bone mineral in adjacent bone is accompanied also by increased accretion. On this point the data conflict.

Bauer and Ray (1968) on the basis of a four-compartment model for strontium kinetics in man were able to simulate external counting curves over the knee and thigh in normal human subjects during five days from the moment of injection of  $\text{Sr}$ . On the basis of a two-compartment model similar simulations were made during the interval 1—10 days following injection of  $\text{Ca}$  or  $\text{Sr}$  in man under normal conditions and in general metabolic bone disease (Wendeberg, 1961).

#### 4 Purpose of this investigation

The purpose of this investigation was to study the mineral metabolism in fractures of the tibial shaft in man by means of external counting of intravenously injected Sr. The tibia was selected for this investigation because it lends itself well to external measurements over the fracture as well as over adjacent bone. The intact leg served as control.

On the basis of a two-compartment model for strontium metabolism the results of the tracer study were interpreted to give information on changes in the accretion rate induced by the fracture.

## A CLINICAL MATERIAL

The present investigation was based on 53 isotope studies in 51 patients with a fracture of the shaft of one tibia. There were 38 males and 13 females ranging in age between 19 and 77 years. One patient without fracture was included for comparison with the fracture cases. Six patients were hospitalized during the investigation period.

With only two exceptions the patients had no other lesions in their lower limbs: one had a fracture of the contralateral patella 4 months prior to the investigation and one had an osteoma of the ipsilateral femoral shaft. The material does not include any cases of fractures through localized bone lesions.

Forty-three fractures were classified as normally healing. At the time of the isotope investigation they were healed or were subsequently found to have healed within six months after fracture. Eight fractures showed delayed or non union and/or osteitis. Pertinent data of the individual cases are given in Table I (normally healing fractures) and Table II (delayed healing, pseudarthrosis and/or osteitis). Case numbers in the following refer to these tables.

In 30 cases the fracture was located in the lower third of the tibia, in 19 cases in the middle third of the tibia and in 2 cases in the upper third of the tibia.

Twelve of the fractures were transverse, 18 fractures were oblique and 21 fractures were comminute. In 11 cases the fracture was open.

The cause of the fractures were traffic accidents (25 cases), falls (14 cases), sport accidents (4 cases) or crush injuries (8 cases).

The fractures were treated by osteosynthesis with Lane's or Egger's plate (17 cases), osteosynthesis with one or more screws (17 cases), osteosynthesis with Rush pin (1 case), sliding tibial graft (2 cases), transfixation (2 cases), and immobilization in plaster only (11 cases). In all of the cases the fractured leg had been immobilized in a plaster cast from groin to toes for a period ranging from 2 to 5 months for the group of normally healing fractures.

The interval between the fracture and the isotope study varied between 2 days and 106 months. Twenty-two patients, selected to observe the later stages of fracture repair, were obtained from a group of cases under follow-up in a clinical investigation to be reported in detail later (Bauer and Widmark, 1962).

## B. METHOD

1 *Dose and administration of Sr* — Each patient received an intravenous injection of 25–50  $\mu\text{C}$  carrier-free  $\text{Sr}^{90}$  ( $\gamma$ -emitter with half-life of 65 days). The amount of radiation delivered to the skeleton by this dose was calculated to be less than the maximal permissible dose recommended by the International Commission on Radiological Protection (Bauer and Wendeborg, 1959).

2 *External counting measurements* — At specific intervals of time after the injection of the isotope, activity measurements were performed with scintillation detectors over various locations of the fractured leg and over corresponding locations of the intact leg. Measurements were made over (a) the thigh, (b) the knee and (c) at 5–7 points 5 cm apart from each other over the tibia. Duplicate measurements were made at each location on each occasion. The detector was repositioned between the two duplicate measurements. In order to ensure exact repositioning of the detector in duplicate measurements and in measurements at various intervals after the injection each area to be measured was marked with red ink. Marks were thus made (a) over the

femoral shaft 20 cm proximal to the base of the patella (b) over the patella and (c) over the fracture and as many points as possible 5 cm apart from one another and on both sides of the fracture

During the external counting the patient was in the supine position with the legs slightly abducted and the patella facing upwards. The legs were held in position by sand bags. The scintillation detector was placed with its outer aperture horizontal and close to the skin over the location marked with red ink. Care was taken to adjust the detector exactly over the mark. For measurements of patients still having plaster this was removed or reduced to a posterior plaster splint.

The examination period covered 14 days. In most cases measurements were made only at 1 week and 2 weeks after the injection, in some also on intermediate occasions. In the latter group then the activities over the fracture and over the corresponding location of the intact tibia were measured every 1 to 5 minutes during the first hour after injection using two similar counting equipments. About 1 hour after the injection, activity measurements were made over the thighs, knees, fracture and intact tibia. The detector used for these measurements was also employed for repeated measurements over a period of 2 weeks after the injection. Measurements over all counting locations of the tibia (scanning) were performed only at 1 and 2 weeks after injection.

The results of the activity measurements are given as

1) *Activity* which means the externally recorded counting rate given as fraction of the counting rate of a Sr standard sample measured under standard conditions. This unit was used to describe the changes with time in counting rate recorded over one and the same location.

2) *Activity ratio* which means the ratio between the counting rate over two anatomically corresponding locations such as the knees. The activity ratio thus differs from unity if the two locations differ from one another in activity. In the following, the abbreviations (a) *thigh/control ratio*, (b) *knee/control ratio* and (c) *fracture/control ratio* will be used for external counting rate ratios (a) thigh of fractured leg/thigh of intact leg, (b) knee of fractured leg/knee of intact leg, and (c) fracture/intact tibia respectively.

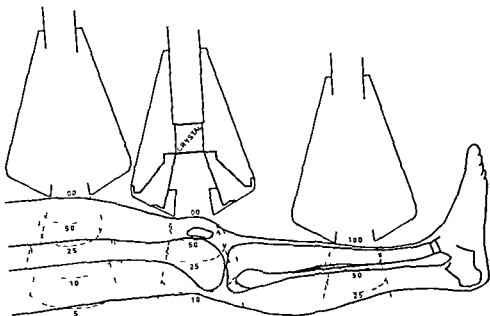


Fig 1 Figure shows isoactivity curves projected over the three main counting locations. The curves were obtained by measurements of the activity of a point source of  $10 \mu\text{C Sr}$  in different positions of a vertical plane through the crystal center. The measurements were performed with the  $\text{Sr}^{88}$  source placed in water assuming the radiation absorption in water being about equal to that in the body tissues. The counting equipment was the same as that used for external counting in the patients. The lower margin of the 12° wide angle collimator was placed at the surface of the water. The figures give the activity as fraction of the activity recorded with the  $\text{Sr}$  source placed centrally in the outer aperture of the collimator. The counting efficiency of the activity retained in bone tissues thus differs between the counting locations. This may be part of the explanation of the different activity curves obtained over the thigh the knee and the tibia and stresses the importance of anatomical identity between counting locations to be compared.



The influence on the external counting rate of anatomical factors *e.g.* the proportions between the amounts of soft tissues and bone tissues and the position of these tissues in regard to the detector is indicated in Fig. 1. It must be realized that even small differences in anatomy between two locations may make the activity ratio deviate from unity. Thus for example atrophy of the thigh muscles in the fractured leg, oedema in the fracture region or increased amounts of bone tissue (callus) under the detector will increase the counting rate over the fractured leg. In the patients studied atrophy of thigh muscles was observed but the difference between the circumference of the thigh in the two legs never exceeded 2 cm. Excessive amounts of callus were not observed in the patients studied. These sources of error must be taken into account in the interpretation of activity ratios which deviate only slightly from unity.

**3 Equipment** — The equipment described by Bauer and Wendeberg (1959) was used in this investigation. Activity measurements were thus made with a lead shielded collimated scintillation detector with a  $1.5 \times 1$ " or a  $2 \times 2$ " NaI(Tl) crystal. A 12° wide angle collimator (diameter of outer aperture 67 mm) was used throughout the investigation and in one case (Case II-6) also a 10 mm slot aperture insert. The distance from the center of the crystal to the outer aperture of the collimator was about 15 cm. The pulses from the photomultiplier were fed into a scaler via a linear amplifier and a pulse height analyzer. With the exception of the measurements made during the first hour after the injection of Sr at least 1 000 impulses were counted on each occasion within a 7.5 or 10 volt discrimination window over the 0.51 MeV energy peak of S.

**4 Statistics** — The counting rate was corrected for background activity and for physical decay of the Sr<sup>85</sup>. The background activity was recorded with the detector placed over the examination table and with the apparatus adjusted as for measurements of the patients. The background activity was about 0.5 and 1.2 counts per second for the  $1.5 \times 1$ " and  $2 \times 2$ " crystals respectively. Correction for physical decay was made by expressing the counting rate over the patient as fraction of the counting rate of a Sr<sup>85</sup> standard. The stability of the apparatus was checked daily by measuring the activity of a Na<sup>22</sup> standard (half life 2.6 years).

The lowest counting rates in this investigation were recorded on measurements over the tibia 14 days after injection of Sr — they were never

found to be less than twice the background activity. This implies that the standard deviation of the single observation due to random decay was less than  $\pm 2\%$ .

Calculated on the basis of 150 duplicate measurements over fractures, knees and thighs, the difference between the two measurements over the same area with intermediate repositioning of the detector was  $2.7 \pm 1.8\%$  of the mean value of the two counting rates.

In 50 patients without local bone lesions in the lower legs, the activity ratios between the two knees at 1 and 2 weeks after injection of Sr were  $1.06 \pm 0.06$  (mean  $\pm$  S.D.) and  $1.09 \pm 0.09$  respectively. The activity ratios between the two thighs at 1 and 2 weeks after injection were  $1.05 \pm 0.06$  and  $1.06 \pm 0.09$  respectively. The activity ratios were calculated by division of the higher counting rate with the lower one.

*a. General mineral metabolic studies* — In four hospitalized patients (Cases I-3, II-4, II-6 and II-7) the urinary and faecal excretion of Sr and the serum activity were determined during a 14-day post-injection period. Based on these data, the accretion rate of the total skeleton, the size of the exchangeable calcium pool in the body, the excretion rate and the body distribution of Sr were calculated according to Wendeborg (1961). The data thus obtained were used for the interpretation of external counting measurements.

## C. RESULTS

### 1. Intact bone

In the subject without fracture (Fig. 2) the activity over the knee rose from the moment of the injection to reach a maximum value at about 24 hours and then gradually dropped throughout the 14-day period studied. The activity over the thigh reached an activity peak as early as 10 minutes after the injection and then decreased continuously throughout the period of the investigation. The activity over the tibia reached an activity maximum within 24 hours of the injection and then decreased. The counting rate over the knee region was higher than over the thigh and tibia and somewhat higher over the thigh than over the tibia. The activity curves given in Fig. 2 are representative for the normal activity pattern observed in a large number of patients with or without metabolic bone disease.

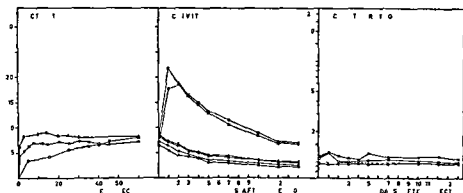


Fig 2 Activity curves obtained by external counting over the thigh ( $\Delta$ ) the knee ( $\square$ ) and the tibia ( $\circ$ ) at various intervals following injection of  $40 \mu\text{C Sr}$  to a normal man aged 42 without bone injury

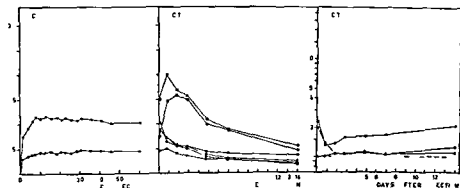


Fig 3 Activity curves in a man aged 37 given  $40 \mu\text{C Sr}$  intravenously two days after he sustained a closed comminute fracture of the middle third of his right tibia (Case 11)

Explanations of symbols for Figs 3—10 and 18—20

- fracture and activity ratio fracture/intact tibia
- intact tibia
- knee of fractured leg and activity ratio knee of fractured leg/knee of intact leg
- knee of intact leg
- ▲ thigh of fractured leg and activity ratio thigh of fractured leg/thigh of intact leg
- Δ thigh of intact leg

## 2 Normally healing fractures

a *Variation in pattern of uptake of Sr with age of fracture* — The activities over the lower limbs during a 14 day period following injection of  $\text{Sr}^{88}$  at increasing intervals of time following fracture of the tibia are shown for 8 patients in Figs 3—10

In a patient, who received  $\text{Sr}^{88}$  2 days after he sustained a fracture of the tibia (Fig 3), the activity recorded over the fracture reached a maximum at about 8 minutes after injection, and thereafter decreased but stayed above that recorded over the control tibia throughout the 14 days of investigation. The fracture/control ratio thus dropped from 2.6 at 10 minutes to 1.3 at Day 1. The fracture/control ratio then increased to 2.1 recorded at Day 14. During the first few days after the injection the activity of the knee of the fractured leg was higher than that of the control knee with a knee/control ratio of 2.0 at 1 hour. This ratio decreased later to about unity. No difference in activity was noted between the two thighs.

The pattern of the activity curves recorded in a patient who received the  $\text{Sr}$  4 days after the fracture (Fig 4) was found to be about the same as that of the patient described above.

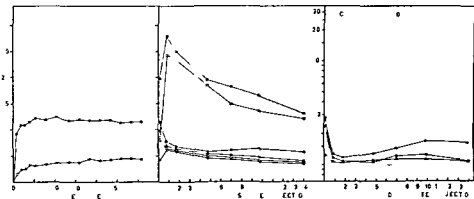


Fig 4 Activity curves in a man aged 19, given  $30 \mu\text{C}$   $\text{Sr}^{88}$  intravenously four days after he sustained a closed transverse fracture of the middle third of his right tibia (Case I-2). The fracture was reduced and immobilized in plaster. For explanation of symbols, see Fig 3.

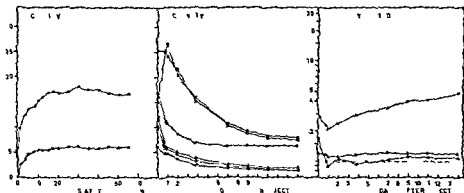


Fig 5 Activity curves in a woman aged 29 given  $40 \mu\text{C Sr}^{90}$  intravenously ten days after she sustained a closed oblique fracture of the middle third of her left tibia (Case I 3). The fracture was reduced and immobilized by an intramedullary Rush pin and plaster. In this case the externally recorded activity curves were simulated on the basis of a kinetical model see Fig 27. For explanation of symbols see Fig 3.

One patient (Fig 5) received an injection of  $\text{Sr}^{90}$  10 days after the fracture. The activity curves obtained over the fractured and the intact legs differed initially. These differences between the knees and thighs disappeared within 24 hours of the injection. The fracture/control ratio decreased during the initial 24 hours and then increased to 4.8 recorded at Day 14.

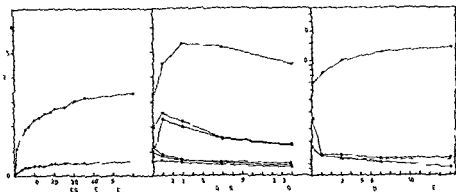


Fig 6 Activity curves in a man aged 19 given  $30 \mu\text{C Sr}^{90}$  intravenously five weeks after he sustained a closed oblique fracture of the distal third of his right tibia (Case I 5). The fracture was reduced and immobilized in plaster. Radiographs of the knee regions in this patient are shown in Fig 39. For explanation of symbols see Fig 3.

Five weeks after the fracture (Fig 6) the pattern of the activity curves differed from those recorded earlier. The activity over the fractured increased for 3—4 days from the moment of injection and then gradually decreased. The fracture/control ratio increased throughout the 14 day-period it was 12.3 at 1 week and 13.6 at 2 weeks after injection. In the knees and in the thighs activity differences were found 1 hour after the injection (knee/control ratio 2.6 and thigh/control ratio 1.3) but not at later intervals.

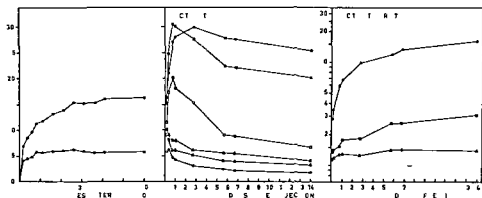


Fig 7 Activity curves in a man aged 19 given  $30 \mu\text{C Sr}$  intravenously five and a half months after he sustained a closed, comminute fracture of the distal third of his right tibia (Case I-7). The fracture was reduced and immobilized by Egger's plate and plaster. This is the same case as in Fig 11. A radiograph of the fractured tibia is shown in Fig 27. For explanation of symbols see Fig 3.

Five and a half months after the fracture (Fig 7) the activity over the fracture increased until the third day after the injection and thereafter decreased. The fracture/control ratio increased throughout the 14 days of investigation, it was 12.8 at 1 week and 16.2 at 2 weeks after the injection. In this case activity differences were recorded also between the two knees and between the two thighs during the entire investigation; the knee/control and thigh/control ratios 14 days after injection were 3.1 and 1.4 respectively.

At 10 months after the fracture (Fig 8) the activity pattern was largely the same as that observed five and a half months after the fracture.

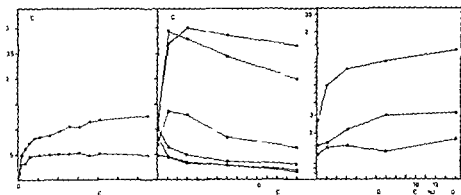


Fig 8 Activity curves in a man aged 32 given  $40 \mu\text{C Sr}$  intravenously *ten months* after he sustained a closed oblique fracture of the middle third of his right tibia (Case I-19) The fracture was reduced and immobilized by Egger's plate and plaster For explanation of symbols see Fig 3

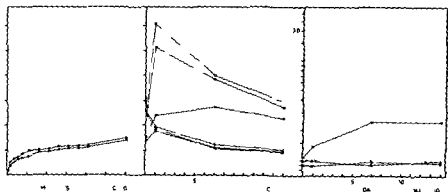


Fig 9 Activity curves in a woman aged 75 given  $50 \mu\text{C Sr}^*$  intravenously *20 months* after she sustained a closed oblique fracture of the distal third of her left tibia (Case I-24) The fracture was immobilized by creos and plaster For explanation of symbols see Fig 3

At 20 months after the fracture (Fig 9) the activity over the fracture region was the same as over the control tibia during the first hour after the injection but later the activity over the fractured tibia was higher than over the normal tibia The fracture/control ratio was 2.4 both 1 and 2 weeks after the injection The activities over the thighs and over the knees were lower in the fractured leg than in the control leg

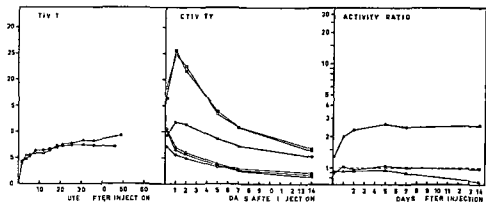


Fig 10 Activity curves in a man, aged 54, given  $50 \mu\text{C Sr}$  intravenously 50 months after he sustained a closed comminute fracture of the distal third of his left tibia (Case I-30). The fracture was immobilized by screws and plaster. This patient was studied also 41 months after fracture, see p 30. For explanation of symbols see Fig 3.

At 55 months after the fracture (Fig 10) the activity pattern was largely the same as that observed at 20 months.

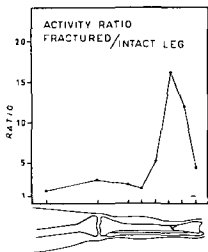


Fig 11 Activity ratios fractured/control leg obtained by external counting two weeks after injection of  $30 \mu\text{C Sr}$  to a man, aged 19 with a  $5\frac{1}{2}$ -month old tibia fracture (Case I-7). A radiograph of the fractured tibia at the time of the isotope investigation is shown in Fig 27. Activity curves of this patient are given in Fig 7.



b *Activity ratios fractured/intact leg 14 days after injection of  $Sr^{90}$*  —

Fig 11 shows the external counting rate ratio fractured/control leg obtained 2 weeks after injection of  $Sr^{90}$  into a patient with a 5½ month-old tibial fracture. The activities in all locations of the fractured leg were higher than in corresponding control locations. The largest difference was noted between the fracture and the normal tibia as shown by the peak in the figure. Similar peaks were noted for all the patients examined with the scanning technique. The peaks tended to vary in height with the age of the fracture at the time of the isotope study (compare Figs 11 and 12; see also Fig 15) and in width with the anatomical type of the fracture

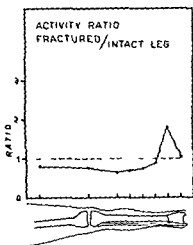


Fig 12 Activity ratios fractured/control leg obtained by external counting two weeks after injection of 40  $\mu C$   $Sr^{90}$  to a woman aged 39 with a 58-month-old tibial fracture (Case 1-33). A radiograph of the fractured tibia at the time of the isotope investigation is shown in Fig 28

(Fig 13). The peaks were thus more broad based when the fractures were spiral than when they were transverse. Additionally, less marked peaks were noted over fibular fractures at levels other than that of the tibial fracture. An increased activity uptake was observed also over the donor site of a tibial graft proximal to the fracture (Fig 14).

The activity ratios recorded 2 weeks after the injection are summarized in Fig 15. The fracture/control ratio was above unity in all patients studied. It increased rapidly during the first few months after the fracture (Fig 15 insert). The highest values were recorded about 6–8 months after the fracture: activity ratios fracture/control up to 30 were observed with a mean of  $15.0 \pm 7.2$  for 14 patients studied at 0–10 months after the fracture. With increasing time after the fracture the ratios decreased. However, in the ten patients studied more than five years after fracture the fracture/control ratio still was significantly higher than unity (mean value  $2.0 \pm 0.9$ ).

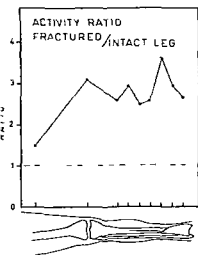


Fig 13 Activity ratios fractured/control leg obtained by external counting two weeks after injection of 50  $\mu$ C Sr to a man aged 35, with a 15-month old tibial fracture (Case I-23) There was a comminute fracture of the distal third of tibia and a longitudinal fracture through the middle third of tibia Fibula was fractured through its upper third Radiographs of the fractured leg after injury, after reposition and fixation with screws and at the time of isotope investigation are shown in Fig 29

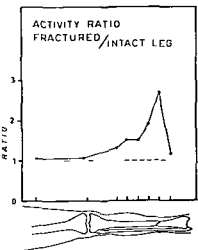


Fig 14 Activity ratios fractured/control leg obtained by external counting two weeks after injection of 40  $\mu$ C Sr to a woman aged 46 with a 64-month old tibial fracture primarily treated by sliding bone graft operation (Case I-35) Note the broad peak including the donor site of the graft A radiograph of the fractured tibia at the time of the isotope investigation is shown in Fig 30

No significant differences were observed between the fracture/control ratios obtained over fractures of the proximal, middle and distal third respectively of the tibia

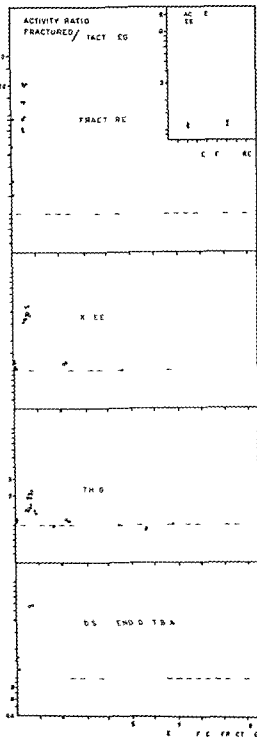


Fig 1> Activity ratios fractured/intact leg obtained two weeks after injection of Sr. The figure includes activity ratios obtained one week after injection for three patients (Cases I 20 II 6 and II-7) who were not measured later. Knee/control and thigh/control ratios have been omitted for two patients who suffered from a fracture of the patella and an osteoma of the femoral shaft respectively (Cases I-6 and I 32). The activity ratios given for the distal end of the tibia include only cases with fractures of the middle or proximal third of the tibia to exclude influence from activity in the fracture region.

- normally healing fractures
- pseudarthrosis delayed healing or osteitis

During the first month after the fracture the *knee/control ratio* was normal. Thereafter it increased to a peak about 6 months after the fracture with a mean of  $3.8 \pm 1.3$  for 14 patients studied at 5—10 months after fracture. It then decreased at roughly the same rate as the fracture/control ratio. Up to 4—5 years after the fracture the *knee/control ratio* was *above* unity and later *below* unity.

The *thigh/control ratio* varied with time in roughly the same way as the *knee/control ratio*; the peak activity ratios were lower, however. The mean of the activity ratios for 14 patients studied at 5—10 months after fracture was  $1.6 \pm 0.5$ . During the first 2—3 years after the fracture the *thigh/control ratio* was *above* unity and later *below* unity.

The activity ratios obtained over the *distal tibia* are given in Fig. 15 only for patients with fractures of the middle or proximal third of tibia in order to exclude any influence of activity in the fracture region on the measurements. The change in the activity ratio with time after the fracture was found to be largely the same as that recorded over the knee region.

Two patients were examined twice. One of those (Case I-4) was studied 1 month and 8 months after the fracture. Between the two investigations the fracture/control ratio at Day 11 decreased from 9.1 to 8.3; the knee/control ratio increased from 1.2 to 4.5; and the thigh/control ratio increased from 1.1 to 1.5. The other patient (Case I-30) was examined 41 and 50 months after the fracture. During this interval the fracture/control ratio at Day 14 decreased from 7.7 to 2.5; the knee/control ratio from 1.3 to 1.0; and the thigh/control ratio from 1.0 to 0.70. The variation in the activity ratios with time after fracture in these two patients thus fitted the pattern observed for the material as a whole.

Fig. 16 Activity ratios recorded at two weeks in per cent of those recorded at one week following intravenous injection of Sr. For explanation of symbols see Fig. 15.

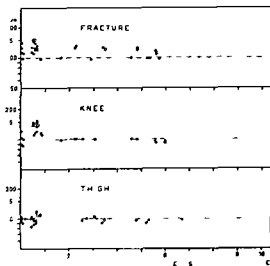


Fig 16 gives the activity ratios obtained two weeks after injection in per cent of the activity ratios obtained one week after injection. In most cases the fracture/control ratios rose between the two measurements. This was also noted for the knee/control ratios and the thigh/control ratios during the first years after fracture. Later when most of these ratios were below unity they tended to drop from 1 to 2 weeks after injection.

### 3 Fractures with delayed or non union

Eight patients had pseudarthrosis, delayed healing and/or osteitis. Clinical data and radiographs of the fractures of these patients are given in Table II and Figs 31—38.

Fig 17 Activity ratios fractured/control leg obtained by external counting two weeks after intravenous injection of 50  $\mu$ C Sr to a man aged 58 with six month old slowly uniting fractures of his tibia and fibula (Case II-1). Radiographs of these fractures at the time of the isotope investigation are shown in Fig 31.

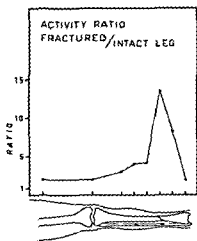


Fig 17 gives activity ratios fractured/control leg obtained two weeks after injection of Sr in Case II-1. A marked peak was found over the fracture of the tibia and a less marked peak was found over the non-uniting fracture of the fibula.

Figs 18 and 19 show the activity curves for two patients (Cases II-3 and II-4) with delayed healing of fractures of the tibia. The fractures were 7 and 8½ months old respectively at the time of investigation. The activity patterns in these cases did not differ from those obtained in normally healing fractures of corresponding age.

For one patient with a 26-month-old tibial pseudarthrosis (Case II-6) the activity pattern was largely the same as those of normally healing fractures of corresponding age (Fig 20—A). Eight days after the injection of Sr the lower leg of this patient was amputated. Immediately before

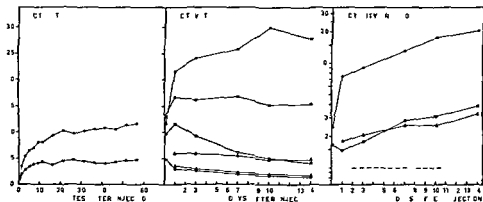


Fig 18 Activity curves in a man aged 22 given  $40 \mu\text{C Sr}^{90}$  intravenously 7 months after he sustained an open transverse fracture of the middle third of his right tibia (Case II-3). The fracture was treated primarily with Egger's plate and plaster. At the time of the isotope injection he had delayed healing caused by infection. Radiographs of his tibia are shown in Fig. 33. For explanation of symbols see Fig. 3.

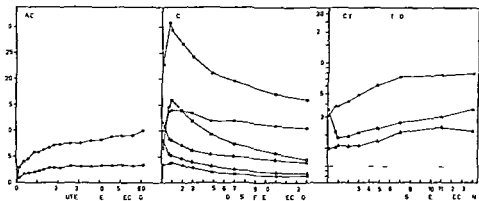


Fig 19 Activity curves in a man, aged 38 given  $40 \mu\text{C Sr}^{90}$  intravenously  $8\frac{1}{2}$  months after he sustained an open comminute fracture of the middle third of his left tibia (Case II-4). At the time of the isotope investigation he had delayed healing of the fracture. Radiographs of his tibia are shown in Fig. 34. For explanation of symbols see Fig. 3. In this case the externally recorded activity curves were simulated on the basis of a kinetical model see Fig. 24.

the amputation scanning was performed over the fracture and its surroundings and over the intact tibia with a 10 mm slot aperture insert. These measurements showed a well defined activity peak over the tibial fracture and a less marked peak over the fibular fracture (Fig. 20—B). The activity

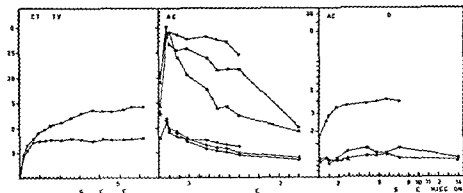


Fig 20—A Activity curves in a man aged 58 given 50  $\mu$ C Sr intravenously 26 months after he sustained an open transverse fracture of the middle third of his right tibia (Case II 6). Because of pseudarthrosis a lower leg amputation was decided on. The isotope injection was made 8 days prior to amputation. Immediately before amputation scanning with a 10 mm slot aperture insert was performed over the fracture adjacent parts of tibia and fibula and over the intact left leg (in vivo of Fig 20—B). After the amputation the tibia was dissected free from soft tissues and the fibula cut transversely into 10 mm slices which were ashed dissolved in nitric acid and individually counted in a well scintillation counter (in vitro of Fig 20—B). The most active slice included the pseudarthrosis and contained 0.2 % of the given dose of Sr (see Table III). A radiograph of the pseudarthrosis is shown in Fig 36. For explanation of symbols see Fig 3.

ratio fracture/intact tibia was 5.8. After the amputation the tibia was dissected free from soft tissues and from the fibula and sawn into 10 mm thick slices corresponding to the areas covered by the collimator during external measurements. Measurements were then performed with the same equipment as used for in vivo measurements with the bone slice containing the pseudarthrosis placed vertically and centrally under the 10 mm slot aperture insert and with its upper margin 1 cm from the outer aperture of the insert. The counting rate recorded over this single bone piece (1.9 counts/second) was lower than the in vivo counting rate over the pseudarthrosis (2.6 counts/second). By adding the two bone slices adjacent to the pseudarthrosis the counting rate was about 25 % higher than earlier (2.4 counts/second) but still somewhat lower than in in vivo measurements (fibula was removed). No further increase in the counting rate was observed by placing more bone pieces at the side of the others. Thereafter the bone slices were ashed and dissolved in nitric acid and the activity

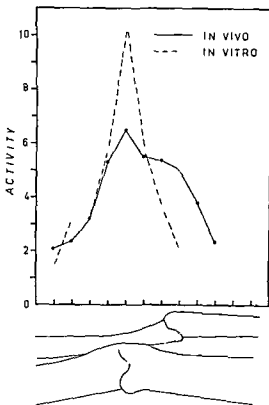


Fig 20—B See legend of Fig 20—A

was determined by measurements in a well scintillation counter (Table III and Fig 20—B). The activity of the pseudarthrosis area was found to be higher than that of adjacent bone. Agreement was thus found between measurements made *in vivo* and *in vitro*.

The activity ratios obtained 2 weeks after the injection in the poorly healing cases are given in Fig 15 together with those obtained in normally healing fractures. The activity ratios were of the same order in both groups. Thus no significant differences in the uptake of  $\text{Sr}^{88}$  was found between fractures in which healing was complicated and those in which healing was normal.

TABLE III

| Distance from pseudarthrosis (mm) | 50 proximal | 40    | 30    | 20    | 10    | 0     | 10    | 20    | 30    | 40    | 50 distal |
|-----------------------------------|-------------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-----------|
| Activity (% of dose)              | 0.027       | 0.033 | 0.040 | 0.070 | 0.111 | 0.200 | 0.112 | 0.061 | 0.060 | 0.025 | 0.018     |
| Ash weight (g)                    | 1.73        | 1.77  | 1.80  | 2.22  | 1.29  | 1.27  | 1.39  | 1.51  | 1.79  | 1.54  | 1.16      |

Absolute activities and ash weights of 10 mm bone slices of tibia obtained from case II 6 after lower leg amputation because of a pseudarthrosis. Fifty  $\mu\text{C}$  of  $\text{Sr}^{88}$  was given intravenously 8 days before operation. See also legend of Fig 20 A.



## A KINETICS OF STRONTIUM IN MAN

In the interpretation of external measurements of Sr kinetical anatomical counting-geometrical and technical factors must be taken into account (Bauer and Wendeborg 1959)

The kinetics of strontium in man was described by Bauer and Ray (1958) on the basis of an open four-compartment system drained by excretion and accretion (Bauer Carlsson and Lindquist, 1955 b 1961) (Fig 21). The rate of exchange between three of the four compartments was so rapid that within about two hours of the injection of  $\text{Sr}^{88}$  they behaved kinetically largely as a single compartment (Figs 21 and 22). After this time the kinetical model may be described as an open two-compartment system where one of the compartments (*S*) represents strontium in plasma, extra- and intracellular fluid and probably also a rapidly exchangeable fraction of bone mineral and where the other compartment (*E*) represents a less rapidly exchanging fraction of the bone mineral strontium.

After intravenous injection of Sr the isotope is thus according to this concept (a) mixed among the different compartments by *exchange* processes (b) deposited in bone by irreversible *accretion* or (c) lost from the body by *excretion* with urine and faeces. Calculated on the basis of this model as described by Wendeborg (1961) the distribution of  $\text{Sr}^{88}$  during a 14 day post-injection period is shown for two patients (cases I 3 and

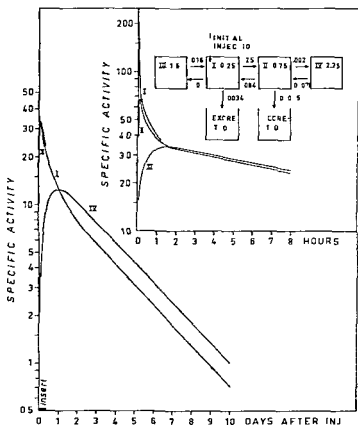


Fig 21 Graph showing the kinetical four-compartment model for strontium metabolism given by Bauer and Ray (1958) and the specific Sr activities in the different compartments during a ten day post injection period. The activity values were calculated by the electronic computer SMIL of Lund University using the values of the sizes of the compartments and the fractional removal rates given by Bauer and Ray. By definition compartment I represents intravascular strontium. Compartment IV was found to represent exchangeable strontium of the bone mineral. Compartments II and III were found to represent strontium of the extravascular body fluids and probably also represent a minor, rapidly exchangeable fraction of bone mineral strontium. Figures within the boxes indicate compartment sizes in units of g Ca. Bauer and Ray used 2.5 liters of plasma as unit. The fractional removal rates (units per minute) are indicated in the model. The figure shows that a few hours after injection compartments I, II and III may be treated as a single compartment.

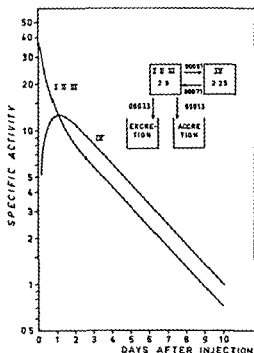


Fig 22 Graph showing the two-compartment model used for the analysis of external counting curves in this study and the specific Sr activities in the two compartments during a ten day post-injection period. The size of the compartments and the fractional removal rates were derived from the four-compartment model given in Fig 21 by lumping of compartments I, II and III. These three compartments are later referred to as compartment S and compartment IV is referred to as compartment E. The specific activity curves were calculated by the electronic computer SMIL of Lund University, Lund. A comparison of the curves given in this figure with those given in Fig 21 reveals that a few hours after injection no differences in specific activity are obtained between the two models. This stresses the validity of the two-compartment model (Wendeborg 1961).

II-4) in Fig 23. The activity in the S compartment dropped continuously after the injection. The activity in the E compartment increased to a maximum about 24 hours after the injection and then decreased almost linearly with the plasma activity. The activity in the accretion and excretion fractions increased cumulatively with the time after the injection.

It should be pointed out that the model used for this analysis does not take into account resorption of labelled strontium incorporated into the skeleton by accretion during 14 days following injection of the tracer.

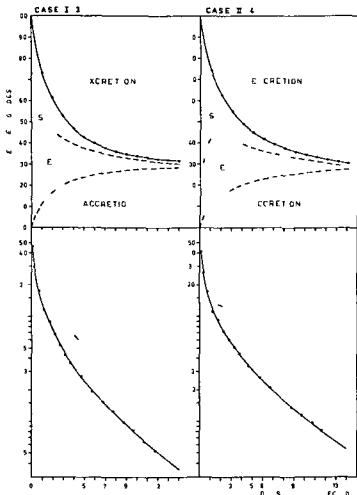


Fig 23 Distribution of Sr and specific activities in compartments S and E ( $s^*$  and  $e^*$ , respectively in per cent of dose/g Ca) during a 14 day period after single intravenous injection of  $\text{Sr}^{88}$  to Case I-3 and Case II-4. The black dots indicate experimentally determined serum activity values. The calculation of the distribution was based on the two-compartment model given in Fig 22 and was made as follows:

*Excretion* by direct measurement of urine and faeces

*Accretion* is the accretion rate times the integrated specific serum activity

$S$  (amount of activity in compartment S) is the size of compartment S times the specific serum activity ( $s^*$ )

$E^*$  (amount of activity in compartment E) is the size of compartment E times specific activity in compartment E ( $e^*$ )

The accretion rate, the sizes of compartments S and E, and the specific activity in compartment E were calculated according to Wendeborg (1961)

|           | Accretion rate<br>(g Ca/day) | S<br>(g Ca) | E<br>(g Ca) |
|-----------|------------------------------|-------------|-------------|
| Case I-3  | 0.52                         | 2.2         | 2.6         |
| Case II-4 | 0.45                         | 2.4         | 2.3         |

The reason for this is that at present all available data suggest that the minimum life span of normal bone tissue in man is longer than 14 days (Bauer Carlsson and Lindquist 1961) Frost Villanueva and Roth (1960) found that the turnover time of normal cortical bone is 7–24 years

## B INTERPRETATION OF EXTERNAL COUNTING OF Sr

Bauer and Ray (1958) on the basis of the four-compartment model were able to simulate external counting curves over the knee and thigh in normal human beings during the first 30 days after the injection of Sr

In the present investigation externally recorded activities over the lower

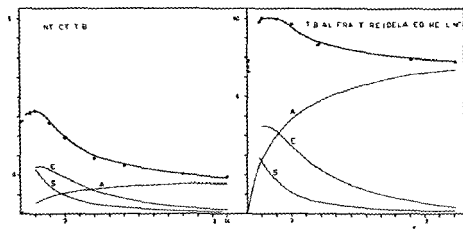


Fig 24—A Graphs representing simulation (on the basis of model shown in Fig 22) of externally recorded activity values over the control right tibia and over the 8½-month-old fracture of the left tibia with delayed healing in Case II-4. Activity curves and radiographs of this case are given in Figs 19 and 34 respectively. Body distribution of Sr after injection is given in Fig 23.

Fractions of the amounts of activity in *S*, *E* and *A* (accretion) in the total body distribution were chosen so that their sum simulated the observed external counting measurements. The ratios of the *S*, *E* and *A* fractions in the fracture over those in the intact tibia were 2.4, 3.7 and 9.2 respectively. The external counting rate ratio fracture/control tibia at Day 14 was 8.0. Note the difference in activity scales in the two graphs.

The observed activity values are shown as black dots.

legs were analysed on the basis of the two-compartment model described above. The total body distribution of Sr in the two cases selected for this analysis is shown in Fig 23. Fractions of the amounts of activity in

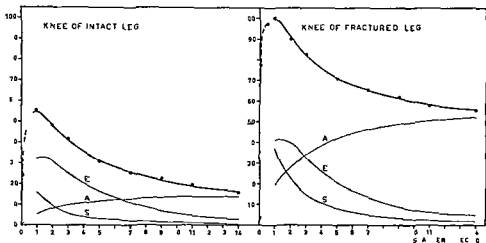


Fig 24—B Graph showing simulation of external counting measurements over knee of intact leg and knee of fractured leg in the same case as in Fig 24—A The ratios of  $S$ ,  $E$  and  $A$  fractions in the knee of the fractured leg over those in the knee of the intact leg were 2.4, 1.3 and 3.7 respectively The external counting rate ratio at Day 14 was 3.6

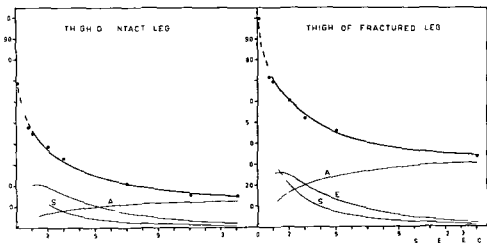


Fig 24—C Graphs showing simulation of external counting measurements over the thigh of the intact leg and the thigh of the fractured leg in the same case as in Fig 24—A The ratios of the  $S$ ,  $E$  and  $A$  fractions in the thigh of the fractured leg over those in the thigh of the intact leg were 1.7, 1.3 and 2.5 respectively The external counting rate ratio at Day 14 was 2.3

compartments *S* and *E* and of the amount of activity accreted (*A*) were chosen so that the sum simulated the externally recorded activities during the interval 1 to 11 days after injection. These simulations are shown in Figs. 24 and 25. All of the curves except the one recorded over the fresh tibial fracture in Case 13 who received the Sr. already 10 days after fracture could be simulated in this way. The cause of the deviation of the fracture curve from the simulated curve may probably be explained by the fact that the accretion rate in a 2—3 weeks old fracture increases rapidly during the investigation period (Fig. 15 insert).

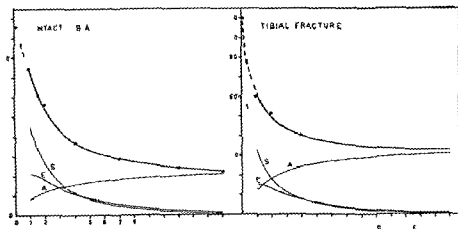


Fig. 25—A Graphs representing simulation of externally recorded activity values over intact left tibia and fractured right tibia. The fracture was ten days old at the time of the isotope injection. The activity curves of this case are also given in Fig. 3. Body distribution of Sr. after injection is seen in Fig. 23. No fit could be found for the fracture curve. The ratios of the *S*, *E* and *A* fractions in the fracture and the intact tibia was 1.1, 1.4 and 3.0 respectively. The external counting rate ratio at Day 14 was 4.8. Note different activity scales in the two graphs.

The observed activity values are shown as black dots.

The strontium in the regions studied — fractures, intact tibia, knees and thighs — would thus be composed of three, kinetically different fractions. The shapes of the activity curves are determined by the relative proportions

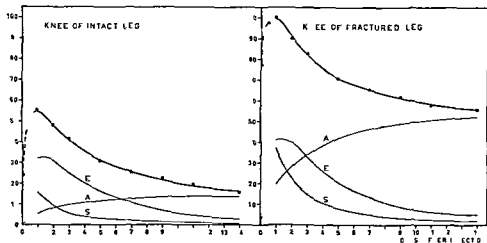


Fig 24—B Graph showing simulation of external counting measurements over knee of intact leg and knee of fractured leg in the same case as in Fig 24—A. The ratios of  $S$ ,  $E$  and  $A$  fractions in the knee of the fractured leg over those in the knee of the intact leg were 2.4, 1.3 and 3.7 respectively. The external counting rate ratio at Day 14 was 3.6.

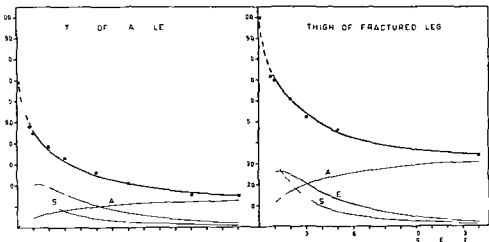


Fig 24—C Graphs showing simulation of external counting measurements over the thigh of the intact leg and the thigh of the fractured leg in the same case as in Fig 24—A. The ratios of the  $S$ ,  $E$  and  $A$  fractions in the thigh of the fractured leg over those in the thigh of the intact leg were 1.7, 1.3 and 2.5 respectively. The external counting rate ratio at Day 14 was 2.3.



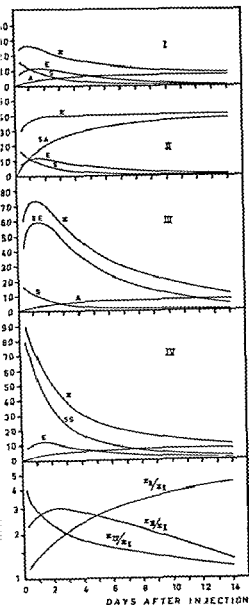


Fig 26—A Graphs showing a theoretical analysis of the shape of activity curves and of activity ratios related to the relative proportions of fractions A E and S of the model shown in Fig 22 Starting from a normal tibia curve (I=Fig 24) the A E and S fractions have separately been increased by a factor of five (II III and IV respectively) In V and VI (Fig 26—B) fractions A and S and A and E respectively have been increased giving activity curves and changes in activity ratios roughly similar to those observed by external counting as shown in Figs 3—5 and 18 respectively

first day or the first few days after the injection and then decreased, and (c) the activity continuously increased during the entire 14-day period studied Examples of (a) were regularly observed over the thigh and over

fresh fractures (Figs 3—5) and are *interpreted* as due to a relatively large fraction of soft tissue activity (Figs 24—C, 25—A and C, and 26, IV and V) Examples of (b) were regularly *observed* over the knee region and in most fractures (Figs 6—10 and 19—20) The activity peak of these curves is *interpreted* as due mainly to a relatively large exchangeable skeletal fraction (Figs 24—A and B 24—B and 26, III) An example of (c) was *observed* in the fracture of Fig 18 and is *interpreted* as an effect of a relatively large fraction of accreted activity (Fig 26, VI)

The change in the external counting rate ratio fractured/intact leg with time after injection is an effect of different proportions of  $A/E$  and  $S$  in the two counting locations compared (*cf* Bauer and Wendeborg 1959) It is clear from Fig 26 that an increase of either  $A/E$  or the  $S$  fraction causes changes in the activity ratios with time after the injection which are characteristic of the fraction that has increased The increase from 1 to 2 weeks following injection observed in most fracture/control knee/control and thigh/control ratios (Fig 16) thus indicates a larger accretion fraction in the fractured leg than in the intact leg Conversely the activity ratios below unity observed over the knee and thigh 5—9 years after the fracture and which decreased between measurements 1 and 2 weeks after the injection suggest a smaller accretion fraction in these locations of the fractured bone than in corresponding locations of the intact bone

The initially increased and then falling activity ratios recorded for cases with fresh fractures suggest an increased soft tissue fraction ( $S$ ) in the fractured leg

It would thus seem that the externally recorded activity curves as well as the change in the activity ratios with time after injection, can be analyzed and interpreted kinetically on the basis of the simplified two-compartment model illustrated in Fig 22

The continuous redistribution of  $Sr^{88}$  from exchangeable to non-exchangeable mineral fractions with time after the injection provides a basis for the interpretation of the activity ratios obtained at different intervals after the injection of the isotope During the earlier intervals after the injection most of the activity injected is distributed in exchangeable compartments ( $S$  and  $E$ ) and only a minor portion has become accreted External counting during the first hour after the injection therefore does not give any direct information of the accretion rate but only of the relative magnitude of the exchangeable compartments under the detector A closer analysis of this part of the curve cannot be made on the basis of the two-compartment model used for analysis of the curves during the later period after

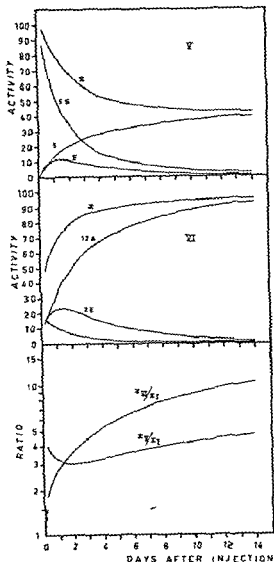


Fig. 26—B See legend of Fig 26—A

the injection (1—14 days) because the model is not valid for short intervals after injection. The activity differences observed between the fractured tibia and the intact tibia during the first hour after the injection and between the knees and thighs of the fractured and the intact leg at measurements about one hour after injection may be ascribed to (a) the hyper-vascularization and the increased blood flow demonstrated in a fractured

leg by angiographic studies in human beings and experimental animals both in normally healing fractures and in pseudarthrosis (Lexer, 1934 Baumgarthl, Gremmel and Willmann, 1958 Judet, Judet and Roy-Camille, 1958, Wray and Lynch, 1959, Gothman, 1960), and (b) the positive correlation between the accretion rate and the size of the exchangeable mineral pool of the skeleton, found in studies of the mineral metabolism of the entire skeleton (Bauer Carlsson and Lindquist, 1955 b, Heaney and Whedon, 1958 Bauer and Wendeborg, 1961) It seems reasonable that the vascular factors predominate in fresh fractures in which accretion is still slow and that the larger exchangeable bone mineral fractions predominate in fractures with a high accretion rate

The rapid uptake of  $\text{Sr}^*$  in the fracture region during the first hour after the injection and the high fracture/control activity ratios observed subsequently both in normally healing fractures and in cases of delayed and non-union indicate an adequate blood supply to the fracture region and a rapid exchange of mineral between body fluids and the callus These studies, as well as earlier experimental isotope studies of fracture healing thus provide evidence that the humoral source of bone salt is essential for the normal progress of healing as claimed by Urist (1942) It is seen in Fig 20—B that the major part of the activity recorded by external counting over a fracture really is located in the actual region of the fracture

Fourteen days after the injection most of the activity retained in the body has been incorporated in the skeleton by accretion (Fig 23) The ratio between two externally recorded activities may then be interpreted as an index of the difference in the amount of  $\text{Sr}^*$  accreted within two body locations measured The amount of activity accreted is a function of the accretion rate and the concentration of tracer in the serum (Bauer, Carlsson and Lindquist 1955 b 1961) The concentration of  $\text{Sr}^{**}$  in the serum may be regarded as equal in both legs The activity ratios at Day 14 may thus be interpreted as indices of differences in accretion rates (Bauer and Wendeborg, 1959) For two reasons, however, these differences are not absolute but relative (a) the activity recorded by external counting 14 days after the injection of  $\text{Sr}^*$  is not exclusively accreted activity but contains a minor fraction of exchangeable activity (Figs 24 and 25) and (b) a wide angle collimator was used in this investigation

The size of the collimator is of importance mainly for measurements over skeletal lesions which are small in relation to the aperture of the collimator such as fractures The use of a wide-angle collimator will then give a lower activity ratio than will a narrow-angle collimator because bone tissue of a high specific activity in the actual fracture region, seen by

the detector is diluted with bone tissue of a lower specific activity adjacent to the fracture (Bauer and Wendeberg 1959). These two factors imply that the activity ratio observed 14 days after the injection is somewhat smaller than the absolute difference in the accretion rate between the two areas studied.

The external counting technique has been used by MacDonald (1958) for studies of tibial fractures in rabbits. By external counting of Na and R1 SA an increased vascularity was demonstrated in the fractured leg. External counting curves recorded over tibial fractures in rabbits during the first hour after injection of  $\text{Sr}^{90}$  are similar to those found for human beings in this study. Thus the activity peak was reached earlier in fresh fractures than in older fractures.

MacDonald (1958) also made external counting over fractured and normal tibias in rabbits up to 100 days after parenteral injection of  $\text{Sr}$ . During later intervals of time the  $\text{Sr}$  activity in the fracture decreased at a faster rate than did that in the normal control tibia presumably because of the progressive resorption of labelled callus. In the present investigation activity measurements were made only during 14 days after injection of  $\text{Sr}^{90}$ . During this interval the activity in the normal tibia decreased at a faster rate than did that in the fracture. However it is evident that at later intervals of time the activity ratio fractured/intact leg will decrease because the life-span of the mineral in the fracture region is shorter than that in normal bone.

## C CHANGES IN THE ACCRETION RATE INDUCED BY THE FRACTURE

### 1 Accretion rate in the fracture region

*a Normally healing fractures* — An increased accretion rate in the fracture region was observed as early as within a week of the fracture (increasing fracture/control ratio from Day 1 after injection of  $\text{Sr}$  in Case I 1). The accretion rate increases rapidly during the first months it is high during the first year after the fracture and then decreases gradually. The most rapid accretion rate in the fracture region was observed 6–8 months after fracture and was found to be up to 30 times that in the normal tibia. For 14 patients studied 5–10 months after fracture the mean accretion rate was at least  $10.0 \pm 7.2$  times that in the intact tibia. Signs of increased bone formation in the fracture region were observed in the present investigation as late as 9 years after the fracture. This shows that

leg by angiographic studies in human beings and experimental animals both in normally healing fractures and in pseudarthrosis (Lexer, 1934 Baumgarthl, Gremmel and Willmann, 1958, Judet, Judet and Roy-Camille, 1958 Wray and Lynch, 1959 Gothman, 1960), and (b) the positive correlation between the accretion rate and the size of the exchangeable mineral pool of the skeleton, found in studies of the mineral metabolism of the entire skeleton (Bauer, Carlsson and Lindquist, 1955 b Heaney and Whedon, 1958 Bauer and Wendeberg, 1961) It seems reasonable that the vascular factors predominate in fresh fractures in which accretion is still slow and that the larger exchangeable bone mineral fractions predominate in fractures with a high accretion rate

The rapid uptake of  $\text{Sr}^{90}$  in the fracture region during the first hour after the injection and the high fracture/control activity ratios observed subsequently both in normally healing fractures and in cases of delayed and *non-union* indicate an adequate blood supply to the fracture region and a rapid exchange of mineral between body fluids and the callus These studies as well as earlier experimental isotope studies of fracture healing thus provide evidence that the humoral source of bone salt is essential for the normal progress of healing as claimed by Urist (1942) It is seen in Fig 20—B that the major part of the activity recorded by external counting over a fracture really is located in the actual region of the fracture

Fourteen days after the injection most of the activity retained in the body has been incorporated in the skeleton by accretion (Fig 23) The ratio between two externally recorded activities may then be interpreted as an index of the difference in the amount of  $\text{Sr}^{90}$  accreted within two body locations measured The amount of activity accreted is a function of the accretion rate and the concentration of tracer in the serum (Bauer Carlsson and Lindquist, 1955 b 1961) The concentration of  $\text{Sr}^{90}$  in the serum may be regarded as equal in both legs The activity ratios at Day 14 may thus be interpreted as indices of differences in accretion rates (Bauer and Wendeberg 1959) For two reasons however, these differences are not absolute but relative (a) the activity recorded by external counting 14 days after the injection of  $\text{Sr}^{90}$  is not exclusively accreted activity but contains a minor fraction of exchangeable activity (Figs 24 and 25) and (b) a wide-angle collimator was used in this investigation

The size of the collimator is of importance mainly for measurements over skeletal lesions which are small in relation to the aperture of the collimator such as fractures The use of a wide-angle collimator will then give a lower activity ratio than will a narrow angle collimator because bone tissue of a high specific activity in the actual fracture region, seen by

Thus a rapid accretion rate was noted in non uniting fractures in which ro nitrogen examination indicated that the net increase in mineral content in the region of the fracture was slight. This shows that the rate of bone salt accretion is counterbalanced by a rapid rate of resorption of bone salt in poorly healing fractures. This explanation is supported experimentally by the work of Bohr (1955) who found a lower ash weight in poorly healing fractures than in normally healing fractures.

*c Absolute accretion rate* — In Case II-6 in whom lower leg amputation was performed 8 days after injection of Sr the absolute accretion rate in the fracture region may be calculated. On the basis of excretory and serum activity data the accretion rate of the total skeleton and the size of the exchangeable calcium fraction in the skeleton ( $E$ ) were calculated according to Wendeborg (1961). The accretion rate and the  $E$  fraction were found to be 0.57 g Ca/day and 1.7 g Ca respectively. At Day 8 40 % of the given dose of Sr was calculated to be incorporated in the skeleton by accretion and about 10 % was in the exchangeable fraction. From the 10 mm bone slice including the pseudarthrosis 0.2 % of the dose was recovered (Table III). As the accretion rate and the size of the exchangeable calcium fraction in the skeleton are positively correlated (Bauer, Carlsson and Lindquist 1955b; Heaney and Whedon 1956) it may be assumed from the figures given above that 4/5 of the activity recovered from the bone piece was accreted, i.e. 0.16 % of the dose. These figures thus indicate that 0.4 % of the amount of calcium laid down by accretion in the total skeleton was laid down in the pseudarthrosis, i.e. 0.0023 g Ca/day. Bauer (1954) found that in one-week-old fractures of the femur in rats 0.001 g Ca was laid down per day.

The ash weight of the bone piece was 1.27 g which equals 0.50 g Ca. The turnover time of the bone tissue in the fracture region may thus be calculated to be about 220 days. From P data and bone biopsy (Bauer, Carlsson and Lindquist 1957) the turnover time of bone tissue in the proximal part of tibia in a normal man was calculated to be about 1100 days. From studies with tetracyclines even longer turnover times have been described (Frost 1960). The turnover time of the bone tissue in the pseudarthrosis thus was found to be at least 5 times shorter than that in normal tibial bone. This figure may be compared to the activity ratio fracture/intact tibia obtained 8 days after injection in this case with the 10 mm slot aperture insert which was 5.8 (see page 90). Assuming that the total skeleton of this patient contained 1000—1500 g Ca the mean turnover time of the skeleton was calculated from the accretion rate of 0.57 g Ca/day, to be

1 Fifty-one adult patients with fractures of one tibia were studied by external counting with scintillation detectors over the thighs, the knees and the tibias during a 14 day period after intravenous injection of 25—50  $\mu\text{C}$  Sr. At the time of injection the fractures ranged in age from 2 days to about 9 years. Forty three fractures were classified as normally healing fractures and eight fractures showed delayed or non-union and/or osteitis.

2 The pattern of the activity curves recorded over the fractured leg compared to those recorded over the control leg varied significantly with the age of the fracture.

3 In the *fracture region* as compared to the intact tibia an increased uptake of  $\text{Sr}^{88}$  was observed in all cases studied. In fresh fractures this was most marked during early intervals after injection. The activity ratio fracture/control tibia obtained 14 days after injection rose during the first months after fracture to reach a peak value 6—8 months after fracture. The mean of the 14 Day fracture/control ratios obtained in 14 patients studied 5—10 months after fracture was  $15.5 \pm 7.2$ . The 14 Day activity ratios then subsequently dropped. Even 6—9 years after fracture the counting rate over the fracture was higher than that over the intact tibia.

4 In the *knee region* of the fractured leg as compared to the knee region of the intact leg an increased activity uptake was found some months after fracture. About 6 months after fracture a peak value



was reached. The mean 14 Day activity ratio knee of fractured leg/knee of intact leg for 14 patients studied 3—10 months after fracture was  $3.8 \pm 1.3$ . Thereafter the knee activity dropped to reach values which were equal to or lower than that of the control knee at about 4 years and later after fracture.

In the *thigh* of the fractured leg and the *tibia adjacent to the fracture* the uptake of Sr varied according to a pattern similar to that in the knee region of the fractured leg. The mean 14 Day activity ratio thigh of fractured leg/thigh of intact leg for 14 patients studied 3—10 months after fracture was  $1.6 \pm 0.5$ .

5. No differences in activity uptake were observed between normally healing fractures and fractures showing delayed or non union.

6. The activity curves obtained over the thigh, the knee and the tibia of the fractured and intact legs during the interval 1—14 days after injection of Sr could be simulated on the basis of a two compartment model for the kinetics of strontium in the body.

7. Based on this kinetical analysis the externally recorded Sr activity values may be interpreted as follows:

(a) The activity ratios fractured/intact leg obtained during early intervals after injection are mainly related to differences in the size of the exchangeable mineral spaces under the detector.

(b) The 14 day activity ratio of two anatomically comparable locations may be used as a relative index of the difference in the accretion rate (rate of irreversible deposition of bone mineral) in these locations but is somewhat lower than the absolute difference in the accretion rate.

(c) The bone salt laid down in the fracture callus is derived from the body fluids.

(d) The accretion rate in the fracture region is increased within a week of the fracture. It rapidly increases during the first months after fracture to reach a peak value at 6—8 months after fracture. Thus for the 14 patients studied 3—10 months after fracture the mean accretion rate in the fracture region was at least  $1.5 \pm 7.2$  times that in the control tibia. Thereafter the accretion rate drops but still 6—9 years after fracture the accretion rate in the fracture region may be higher than normal.

(e) The accretion rate in the entire fractured leg is increased some months after fracture. This reaction however appears later, is less marked and disappears earlier than in the fracture region. Thus in the knee and thigh region of the fractured leg the mean accretion rate for 14 patients studied

5—10 months after fracture was at least  $3.8 \pm 1.3$  and  $1.6 \pm 0.5$ , respectively, times that in corresponding locations of the control leg

(f) The traumatic osteopenia is caused by increased resorption and not by decreased accretion

(g) The accretion rate in fractures showing delayed or non-union does not differ from that in normally healing fractures

(h) The turnover time of bone tissue in a 26-month-old pseudarthrosis of the tibia was calculated to be about 220 days, which is at least 5 times shorter than in the normal tibia. The accretion rate in the actual region of the pseudarthrosis was calculated to be  $0.0023 \text{ g C/day}$

## ZUSAMMENFASSUNG

1) Es wurden 31 erwachsene Patienten mit einseitigen Tibiabruchen durch aussere Messung mit Scintillations-Detektoren über den Oberschenkeln den Knieen und den Schienbeinen während eines 14 tagigen Zeitraumes nach der intravenösen Injektion von  $20-50 \mu\text{C Sr}$  untersucht. Zur Zeit der Einspritzung waren die Brüche 2 Tage bis etwa 9 Jahre alt. Dreiundvierzig Brüche wurden als normal heilende bezeichnet, acht zeigten verzögerte oder keine Heilungstendenz mit oder ohne Osteitis.

2) Die Aktivitätskurven, die über dem gebrochenen Bein ermittelt wurden, zeigten im Vergleich zu denen über der Kontroll Extremität einen bemerkenswerten Unterschied entsprechend dem Alter der Frakturen.

3) Über dem Knochenbruch wurde verglichen mit der unversehrten Tibia in allen Fällen eine gesteigerte Aufnahme von Sr beobachtet. Bei frischen Brüchen war diese während der ersten Zeit nach der Injektion am meisten augenfällig. Der Aktivitätsquotient Fraktur-Stelle Kontroll-Tibia ermittelt 14 Tage nach der Injektion von Sr, erhöhte sich während der ersten Monate nach der Fraktur um sechs bis acht Monate danach einen Höchstwert zu erreichen. Der Mittelwert der 14-tägigen Aktivitätsquotienten, der bei der Untersuchung von 14 Patienten während der ersten 5-10 Monate nach der Fraktur ermittelt wurde, war  $15.5 \pm 7.2$ . Danach verminderte sich der Wert der 14-Tage-Quotienten laufend. Auch 6-9 Jahre nach dem Knochenbruch war der Messwert über dem gebrochenen Bein höher als über der unversehrten Tibia.

4) Über dem *Knie* des gebrochenen verglichen mit dem Knie des unversehrten Beines wurde einige Monate nach der Fraktur eine gesteigerte Aktivitätsaufnahme gefunden. Etwa 6 Monate nach dem Bruch liess sich ein Höchstwert erreichen. Der Mittelwert der 14-tägigen Aktivitätsquotienten Knie des gebrochenen Beines Knie der unversehrten Extremität war bei 14 Patienten die 5—10 Monate nach dem Bruch untersucht wurden  $3.8 \pm 1.3$ .

Danach nahm die Knieaktivität ab und erreichte Werte, die gleich denen oder niedriger als diese des Kontrollkniees zu einem Zeitpunkt von vier oder mehr Jahren nach der Fraktur waren.

Im *Oberschenkel* des gebrochenen Beines und in dem der Fraktur benachbarten Teil der Tibia war die Aktivitätsaufnahme von  $Sr^{90}$  etwa die gleiche wie im Knie der gebrochenen Seite. Der Mittelwert der 14-Tage-Aktivitätsquotienten Oberschenkel des gebrochenen Beines Oberschenkel des unversehrten Beines war bei 14 Patienten die 5—10 Monate nach dem Bruch untersucht wurden  $1.6 \pm 0.5$ .

5) Es wurde kein Unterschied in der Aktivitätsaufnahme zwischen normal heilenden und solchen Brüchen beobachtet, die verzögerte oder keine Heilung zeigten.

6) Die Aktivitätskurven die über dem Oberschenkel dem Knie und der Tibia des gebrochenen und des unbeschädigten Beines während eines Zeitraumes von 1—14 Tagen nach der Verabreichung von  $Sr^{90}$  ermittelt wurden, konnten auf der Basis eines Zwei-compartment Modelles der Kinetik von Strontium im Körper vorgetauscht werden.

7) Nach Zugrundelegung dieser kinetischen Analyse können die oberflächlich aufgezeichneten  $Sr^{90}$ -Aktivitätswerte wie folgt gedeutet werden.

a) Die Aktivitätsquotienten gebrochenes intaktes Bein ermittelt während der ersten Zeit nach der Injektion, sind hauptsächlich von Grössenunterschieden der austauschbaren Mineralräume unter dem Detektor abhängig.

b) Der 14-tägige Aktivitätsquotient zweier anatomisch vergleichbarer Stellen kann als relativer Index des Unterschiedes der accretions-Geschwindigkeit (=Geschwindigkeit der irreversiblen Ablagerung von Knochen-Mineralien) an diesen Stellen gebraucht werden ist aber etwas niedriger als der absolute Unterschied der accretions-Geschwindigkeit.

c) Die Knochensalze die im Frakturcallus abgelagert sind stammen aus den Körperflüssigkeiten.

d) Die accretions-Geschwindigkeit des Frakturgebietes vergrössert sich innerhalb einer Woche nach dem Bruch. Sie steigt während der ersten

Monate nach einer Fraktur rasch an um etwa 6—8 Monate danach einen Höchstwert zu erreichen. Somit war bei den 14 Patienten die während 7—10 Monate nach der Fraktur untersucht wurden der Zuwachsanteil des Frakturgebietes mindestens  $1,5 \pm 7,2$  mal grösser als der der Kontrolltibia. Danach nimmt der accretions Geschwindigkeit wieder ab kann aber noch 6—9 Jahre nach der Fraktur im eigentlichen Eruchgebiet noch immer höher als normal sein.

c) Die accretions Geschwindigkeit des unversehrten Teiles des gebrochenen Beines ist einige Monate nach der Fraktur erhöht. Diese Reaktion jedoch tritt erst später in Erscheinung ist weniger augenfällig und verschwindet früher als im Frakturgebiet. Somit war die accretions - Geschwindigkeit im Knie und Oberschenkel des gebrochenen Beines bei 14 Patienten die während 7—10 Monate nach der Fraktur untersucht wurden mindestens  $3,8 \pm 1,3$  beziehungsweise  $1,6 \pm 0,3$  mal grösser als die der entsprechenden Stellen des Kontrollbeines.

f) Die traumatische Osteopenie wird durch erhöhte Resorption und nicht durch verminderten Anbau verursacht.

g) Die accretions - Geschwindigkeit bei Frakturen mit verzögerter oder keiner Heilungstendenz unterscheidet sich nicht von der bei normal heilenden Frakturen.

h) Die Umbauzeit von Knochengewebe bei einer 26 Monate alten Pseudarthrose der Tibia wurde auf etwa 220 Tage geschätzt ein Wert der mindestens einem Fünftel des Wertes der normalen Tibia entspricht. Die absolute accretions - Geschwindigkeit der eigentlichen Pseudarthrose wurde auf etwa 0,0023 g Ca pro Tag geschätzt.

## RESUMÉ

1 Cinquante et une personnes adultes, atteintes de fracture d'un tibia, ont été étudiées par comptage externe avec un détecteur à scintillation. Le comptage a été pratiqué sur le tibia, le genou et la cuisse pendant une période de 14 jours après une injection intra-veineuse de 25 à 50  $\mu$ C de Sr. Au moment de l'injection l'âge des fractures s'étalait sur une période de 2 jours à 9 ans.

Quarante-trois fractures ont été classées comme ayant une consolidation normale et huit fractures ont présenté un retard ou une absence de consolidation ou une ostéite soit isolée surajoutée à l'un ou l'autre des deux premiers cas.

2 L'aspect des courbes d'activité relevées sur le membre fracture comparé à celui des courbes relevées sur la jambe de contrôle a varié de façon significative suivant l'âge de la fracture.

3 Dans tous les cas étudiés, une augmentation du taux de Sr<sup>90</sup> fut observée dans la région de la fracture par rapport au tibia intact. Dans les fractures fraîches, cette augmentation fut la plus marquée durant les intervalles de temps proches de l'injection. Le rapport d'activité tibia fracture/tibia de contrôle obtenu 14 jours après l'injection augmenta durant les premiers mois après la fracture pour atteindre un sommet à 6—8 mois après la fracture. La moyenne des rapports fracture/contrôle au 14<sup>ème</sup> jour après l'injection moyenne obtenue chez 14 cas étudiés de 5 à 10 mois après la fracture fut de  $15.5 \pm 7.2$ . Alors, passe cette période, les

rapports d'activite au 14<sup>ème</sup> jour baisserent. Meme 6 a 9 ans apres une fracture, le taux de comptage sur la fracture fut encore plus éleve que sur le tibia sain

4 Dans la region du genou du membre fracture comparativement à celle du genou du membre intact on trouva une augmentation du taux d'activite quelques mois apres la fracture. Une valeur-sommet fut atteinte environ 6 a 8 mois apres la fracture. La moyenne des rapports d'activite au 14<sup>ème</sup> jour du genou cote fracture/genou cote intact relevee chez 14 cas examines 3 a 10 mois apres la fracture fut de  $3.8 \pm 1.3$ . Ensuite l'activite du genou cote fracture diminua jusqu'a atteindre des valeurs qui furent egales ou inferieures du genou de controle environ 4 ans et plus apres la fracture.

Dans la region de la cuisse du membre fracture et dans les regions tibiales adjacentes a la fracture le taux de  $Sr^{90}$  evolua de la meme façon que dans la region du genou. La moyenne des rapports d'activite au 14<sup>ème</sup> jour apres l'injection de la cuisse cote fracture/cuisse coté-sain relevee chez 14 malades examines 3 a 10 mois apres la fracture fut de  $1.6 \pm 0.5$ .

5 Aucune difference des taux d'activite n'a éte observée entre les fractures consolidant normalement et les fractures montrant un retard de consolidation ou une pseudarthrose.

6 Les courbes d'activite obtenues sur le tibia le genou et la cuisse des membres fractures et intacts pendant la periode de 1 a 14 jours apres l'injection de  $Sr^{90}$  peuvent étre representees en ce qui concerne les mouvements du strontium dans le corps sur la base d'un modele à deux compartiments.

7 Basées sur cette analyse cinetique les valeurs d'activite du  $Sr^{90}$  relevees par comptage externe peuvent étre interpretees comme suit:

a) Les rapports d'activite du membre fracture/membre intact obtenus a des intervalles de temps rapproches apres l'injection ont concerne surtout des differences dans la dimension de l'espace mineral échangeable place sous le detecteur.

b) Le rapport d'activite au 14<sup>ème</sup> jour de deux localisations anatomiquement comparables peut nous servir comme un index relatif des differences dans la vitesse d'accroissement (taux de depot irréversible des sels minéraux dans l'os) de ces localisations mais il est quelque peu inferieur à la difference absolue de la vitesse d'accroissement.

c) Le sel osseux depose dans les cals de fracture provient des fluides du corps.

d) La vitesse d'accrétion" dans la région de la fracture augmente en moins d'une semaine après la fracture. Elle augmente rapidement durant les premiers mois après la fracture pour atteindre une valeur sommet à 6—8 mois après la fracture. Ainsi pour les 14 cas étudiés de 5 à 10 mois après leur fracture, la vitesse moyenne d'accrétion dans la région de la fracture fut au moins  $15.5 \pm 7.2$  fois celle relevée dans le tibia de contrôle.

Puis la vitesse d'accrétion baisse, mais 6 à 9 ans après la fracture elle est encore plus élevée que normal.

e) La vitesse d'accrétion" dans le membre entier côté-fracture augmente quelques mois après la fracture. Cette réaction, cependant, semble plus tardive, est moins marquée et disparaît plus précocement que dans la région même de la fracture. Ainsi dans les régions du genou et de la cuisse du membre fracturé la vitesse moyenne d'accrétion pour 14 cas étudiés sur 5 à 10 mois après la fracture, fut respectivement au moins  $3.8 \pm 1.3$  et  $1.6 \pm 0.5$  fois celui des localisations correspondantes du membre de contrôle.

f) L'ostéopénie traumatique est causée par une augmentation de la résorption et non par une "accrétion" diminuée.

g) La vitesse d'accrétion" dans les fractures montrant un retard ou une absence de consolidation ne diffère pas de celle des fractures consolidant normalement.

h) La durée de vie du tissu osseux dans une pseudarthrose du tibia vieille de 26 mois a été estimée à environ 220 jours, ce qui est au moins 5 fois plus court que dans un tibia normal. La vitesse absolue d'accrétion dans cette pseudarthrose a été calculée être de 0,0023 g Ca/jour.

#### ACKNOWLEDGEMENTS

The author wishes to thank Associate Professor C. E. Froberg, Institute of Mathematics, Lund, for help in using the electronic computer SMIL, and Mrs. Ann Britt Nordvi, Mrs. Elsa Greta Andersson, Mr. Tage Bramstäng and Mr. Nils Sorbrus for technical assistance. Financial support was obtained from grants given to Goran C. H. Bauer, M. D. from the Josiah Macy, Jr. Foundation, New York, and the Knut and Alice Wallenberg Foundation, Stockholm.



## REFERENCES

- (Abbreviations according to International code for the abbreviation of titles of periodicals in World Medical Periodicals Ed 2 World Medical Association New York 1957)
- Bauer GCH (1954) Rate of bone salt formation in a healing fracture determined in rats by means of radiocalcium *Acta orthop scand* 23 169
- Bauer GCH and Carlsson A (1955) Post fracture bone salt resorption studied in rats *Acta orthop scand* 25 83
- Bauer GCH Carlsson A and Lindquist B (1955a) A comparative study of the metabolism of  $\text{Sr}^{90}$  and  $\text{Ca}^{45}$  *Acta physiol scand* 35 56
- Bauer GCH Carlsson A and Lindquist B (1955b) Evaluation of accretion resorption and exchange reactions in the skeleton *Kgl Fysiograf Sällskap Lund Fö h* 25 1
- Bauer GCH Carlsson A and Lindquist B (1957) Accretion rate of bone salt in osteoporosis studied by means of  $\text{P}^{32}$  *Acta med scand* 158 139
- Bauer GCH Carlsson A and Lindquist B (1961) Metabolism and homeostatic function of bone *Mineral Metabolism* F Bronner and C. Comar Editors Vol I (pp 609—676) New York Academic Press
- Bauer GCH and Ray RD (1958) Kinetics of strontium metabolism in man *J Bone Jt Surg* 40 A 171
- Bauer GCH and Scoccianti P (1961) Uptake of  $\text{Sr}^{90}$  in non malignant vertebral lesions in man *Acta orthop scand* (in the press)
- Bauer GCH and Wendeberg B (1959) External counting of  $\text{Ca}^{47}$  and  $\text{Sr}^{90}$  in studies of localised skeletal lesions in man. *J Bone Jt Surg* 41 B 558
- Bauer GCH and Wendeberg B (1961) Twenty four hour plasma Ca concentration as indicator of the metabolic status of the skeleton To be published.

- Bauer GCH and Widmark PH (1962) To be published in *Acta chir scand*
- Baumgarthl F Gremmel H and Willmann KH (1958) Die Durchblutung von frakturierten Unterschenkeln während der Heilung an Hand von arteriographischen Untersuchungen *Zbl Chir Hefte* 28 1386
- Bick EM (1948) Source Book of Orthopaedics 2 edition The Williams and Wilkins Company Baltimore
- Bohr H (1955) Studies on fracture healing *J Bone Jt Surg* 37 A 327
- Bohr H and Sorensen AH (1950) Study of fracture healing by means of radioactive tracers *J Bone Jt Surg* 32 A 567
- Burdeaux B and Hutchinson WJ (1953) Etiology of traumatic osteoporosis *J Bone Jt Surg* 35 A 479
- Cartier PH DeBernard, B and Lagrange J (1956) Studies on the repair of fractures using P<sup>32</sup> *Bone Structure and Metabolism* Ciba Foundation Symposium London
- Copp DH and Greenberg DM (1945) Studies on bone fracture healing I Effects of vitamins A and D *J Nutr* 29 261
- Coste F Layani F Guérin MT and Guérin RA (1959) Fixation osseuse du strontium et du gallium applications cliniques *Rev Rhum* 26 733
- Dow ES and Stanbury JB (1960) Strontium and calcium metabolism in metabolic bone disease *J clin invest* 39 885
- Dudley HC Markowitz HA and Mitchell TG (1956) Studies on the localization of radioactive gallium (Ga<sup>75</sup>) in bone lesions *J Bone Jt Surg* 38 A 627
- Falkenberg J (1961) An experimental study of the rate of fracture healing *Acta orthop scand* Suppl 50
- Falkenheimer M (1955) Bone healing in rats using radioactive calcium *Fed Proc* 14 201
- Frost HM Villanueva AR and Roth H (1960) Measurement of bone formation in a 57 year old man by means of tetracyclines *Henry Ford Hosp Bull* 8 239
- Gothman L (1960) Arterial changes in experimental fractures of the rabbit's tibia treated with intramedullary nailing A microangiographic study *Acta chir scand* 120 289
- Heaney RP and Whedon GD (1958) Radiocalcium studies of bone formation rate in human metabolic bone disease *J clin Endocr* 18 1246
- Judet R Judet J and Roy Camille R (1958) La vascularisation des pseudarthroses des os longs d'après une étude clinique et expérimentale *Rev Orthop* 44 381
- Karsher H (1953) Der Calcium und Phosphorstoffwechsel bei der normalen und gestörten Knochenbruchheilung sowie in frischen und konservierten Transplantaten (Ein Nachweis mit den radioaktiven Isotopen P<sup>32</sup> und Ca<sup>45</sup>) *Arch klin chir* 275 1
- Lacroix P and Ponlot R (1956) Remarques sur l'histopathologie de l'ostéoporose posttraumatique *Acta chir belg* 55 Supplement I 149
- Lexer E (1934) *Lehrbuch der allgemeinen Chirurgie zum Gebrauche für Ärzte und Studierende* 20 Aufl 2 Band Stuttgart Ferdinand Erke

- MacDonald N.S (1958) Kinetic studies of skeletal metabolism by external counting of injected isotopes. The radioisotope osteogram. *J Lab clin Med* 52 541
- MacDonald N.S Lorick P.C and Petriello L.I (1957) Healing bone fractures and simultaneous administration of radioisotopes of sulfur calcium and strontium. *Amer J Physiol* 191 185
- Roche J and Mourgue M (1939) Recherches sur l'ossification VI Modifications générales de la composition des os longs après fracture expérimentale d'une pièce squelettique et unite physiologique de système osseux. *Bull Soc Chim biol* 21 143
- Spencer H Laszlo D and Brothers M (1957) Strontium and calcium metabolism in man. *J clin Invest* 36 660
- Urist M.R (1942) Calcification and ossification III The role of local transfer of bone salt in the calcification of the fracture callus. *J Bone Jt Surg* 24 47
- Urist M.R Mazer R and McLean P.C. (1954) The pathogenesis and treatment of delayed union and non union. A survey of eighty five ununited fractures of the shaft of the tibia and one hundred control cases with similar injuries. *J Bone Jt Surg* 36-A 931
- Wendeberg B (1961) Bone salt accretion rate and exchangeable calcium spaces in man studied with  $Ca^{45}$  and  $Sr^{90}$  I Method of calculation To be published in *Acta med scand*
- Wendeberg B (1962) In preparation
- Wilkinson G.W and Leblond C.P (1951) The deposition of radiophosphorus in fractured bones in rats. *Surg Gynec Obstet* 97 143
- Wray J and Lynch C (1959) The vascular response to fracture of the tibia in the rat. *J Bone Jt Surg* 41 A 1143





# *Normally healing fractures*

| Case no. | Age (years)    | Sex | Type and localization of tibial fracture | Cause of injury                         | Treatment(s) | Plaster (months) | Dose of $^{45}\text{Ca}$ ( $\mu\text{C}$ ) | Months between fracture and isotope injection | Activity ratios   |        |         |         |                |     |     |
|----------|----------------|-----|--|---|--------------|------------------|--|---|-------------------|--------|---------|---------|----------------|-----|-----|
|          |                |     |  |   |              |                  |  |   | Fracture/ control | 1 week | 2 weeks | 3 weeks | Thigh/ control |     |     |
| 1        | 3              | 37  | M  | Closed comminute middle third of tibia  | Fall         | Screws           | 3  | 40  | /s                | 17     | 21      | 11      | 13             | 11  | 11  |
| 2        | 4              | 19  | M  | Closed transverse middle third of tibia | Sport        | Plaster          | 2½   | 30  | /s                | 14     | 16      | 12      | 11             | 11  | 11  |
| 3        | 5 23<br>25 A-C | 29  | F  | Closed oblique middle third of tibia    | Traffic      | Rush pin         | 2½   | 40  | o/                | 34     | 48      | 10      | 11             | 12  | 12  |
| 4        |                | 58  | M  | Closed transverse middle third of tibia | Fall         | Transfixation    | 5  | 50  | 1                 | 92     | 91      | 14      | 12             | 11  | 11  |
|          |                | 59  | M  | Closed transverse middle third of tibia | Fall         | Transfixation    | 5  | 50  | 8                 | 63     | 83      | 33      | 45             | 13  | 15  |
| 5        | 6 39           | 19  | M  | Closed oblique distal third of tibia    | Traffic      | Plaster          | 3  | 30  | 1½                | 123    | 136     | 10      | 10             | 094 | 084 |
| 6        |                | 52  | M  | Closed oblique distal third of tibia    | Traffic      | Transfixation    | 3½   | 50  | 2½                | 145    | 192     | 068     | 076            | 090 | 090 |
| 7        | 7 11<br>27     | 29  | M  | Closed comminute distal third of tibia  | Crush        | Egger's plate    | 3½   | 30  | 5½                | 128    | 162     | 26      | 31             | 14  | 14  |
| 8        |                | 25  | M  | Closed transverse middle third of tibia | Sport        | Plaster          | 3¼   | 30  | 6                 | 64     | 73      | 39      | 56             | 27  | 28  |
| 9        |                | 53  | M  | Closed oblique proximal third of tibia  | Fall         | Egger's plate    | 2½   | 50  | 6                 | 61     | 92      | 24      | 33             | 092 | 074 |
| 10       | 40             | 26  | M  | Closed transverse middle third of tibia | Traffic      | Egger's plate    | 4½   | 25  | 6½                | 47     | 69      | 38      | 56             | 11  | 11  |
| 11       |                | 39  | M  | Closed oblique distal third of tibia    | Traffic      | Screws           | 5  | 50  | 6½                | 190    | 210     | 40      | 44             | 17  | 17  |

|    |    |   |  |         |              |    |    |    |     |     |     |     |     |     |
|----|----|---|--|---------|--------------|----|----|----|-----|-----|-----|-----|-----|-----|
| 12 | 37 | M | Closed transverse<br>middle third of tibia | Traffic | Plaster      | 4  | 35 | 7  | 236 | 300 | 29  | 34  | 22  | 21  |
| 13 | 74 | M | Closed comminute<br>distal third of tibia  | Traffic | Screws       | 4  | 50 | 7  | 69  | 97  | 29  | 37  | 16  | 14  |
| 14 | 33 | M | Open oblique<br>distal third of tibia      | Traffic | Eggers plate | 2  | 40 | 8  | 205 | 252 | 25  | 37  | 23  | 21  |
| 15 | 35 | M | Closed oblique<br>distal third of tibia    | Traffic | Lane's plate | 4½ | 35 | 8  | 164 | 209 | 50  | 60  | 16  | 19  |
| 16 | 53 | M | Closed comminute<br>distal third of tibia  | Crush   | Screws       | 4  | 50 | 8  | 124 | 150 | 25  | 38  | 17  | 16  |
| 17 | 19 | M | Closed comminute<br>middle third of tibia  | Traffic | Eggers plate | 3  | 25 | 10 | 211 | 205 | 13  | 12  | 13  | 14  |
| 18 | 38 | M | Closed comminute<br>middle third of tibia  | Crush   | Eggers plate | 2½ | 50 | 10 | 107 | 125 | 19  | 22  | 11  | 12  |
| 19 | 32 | M | Closed oblique<br>middle third of tibia    | Sport   | Eggers plate | 3  | 40 | 10 | 102 | 136 | 29  | 32  | 13  | 17  |
| 20 | 50 | M | Closed oblique<br>distal third of tibia    | Traffic | Eggers plate | 5  | 50 | 10 | 60  | 22  |     |     | 13  |     |
| 21 | 26 | M | Closed oblique<br>middle third of tibia    | Sport   | Eggers plate | 4½ | 35 | 12 | 59  | 64  | 26  | 37  | 12  | 14  |
| 22 | 41 | M | Open comminute<br>distal third of tibia    | Traffic | Screws       | 4½ | 50 | 14 | 142 | 157 | 25  | 26  | 21  | 22  |
| 23 | 35 | M | Closed comminute<br>distal third of tibia  | Crush   | Screws       | 2½ | 50 | 15 | 27  | 36  | 24  | 31  | 14  | 15  |
| 24 | 75 | F | Closed oblique<br>distal third of tibia    | Fall    | Screws       | 2½ | 50 | 20 | 24  | 24  | 096 | 094 | 090 | 097 |
| 25 | 45 | M | Closed transverse<br>distal third of tibia | Crush   | Eggers plate | 3  | 50 | 27 | 38  | 48  | 12  | 12  | 11  | 11  |
| 26 | 21 | M | Closed transverse<br>middle third of tibia | Traffic | Plaster      | 4  | 30 | 27 | 27  | 35  | 13  | 13  | 10  | 11  |
| 27 | 22 | M | Open transverse<br>distal third of tibia   | Traffic | Plaster      | 3  | 35 | 31 | 23  | 26  | 10  | 10  | 095 | 091 |
| 28 | 27 | M | Closed comminute<br>distal third of tibia  | Traffic | Plaster      | 2½ | 50 | 33 | 30  | 36  | 10  | 11  | 11  | 11  |

TABLE I (continued)

| Case No. | Age (years) | Sex | Type and localization of tibial fracture | Cause of injury | Treatment            | Plaster (months) | Dose of $\text{Sr}^{85}$ ( $\mu\text{C}$ ) | Months between fracture and isotope injection | Activity ratios  |         |              |         |               |         |
|----------|-------------|-----|--|-----------------|----------------------|------------------|--|---|------------------|---------|--------------|---------|---------------|---------|
|          |             |     |  |                 |                      |                  |  |   | Fracture/control |         | Knee/control |         | Thigh/control |         |
|          |             |     |  |                 |                      |                  |  |   | 1 week           | 2 weeks | 1 week       | 2 weeks | 1 week        | 2 weeks |
| 29       | 50          | F   | Closed oblique distal third of tibia     | Fall            | Screws               | 3                | 50   | 35  | 26               | 25      | 12           | 13      | 070           | 083     |
| 30       | 54          | M   | Closed comminute distal third of tibia   | Fall            | Eggers plate         | 5½               | 50   | 41  | 45               | 57      | 12           | 13      | 11            | 10      |
|          |             |     |  | Fall            | Screws               | 5½               | 50   | 55  | 25               | 25      | 10           | 10      | 090           | 070     |
| 31       | 48          | F   | Closed oblique distal third of tibia     | Fall            | Screws               | 1                | 50   | 53  | 22               | 24      | 11           | 11      | 10            | 10      |
| 32*      | 42          | F   | Closed comminute distal third of tibia   | Fall            | Screws               | 3½               | 50   | 58  | 27               | 34      | 14           | 11      | 23            | 30      |
| 33       | 39          | F   | Closed oblique distal third of tibia     | Fall            | Screws               | 4                | 40   | 58  | 15               | 18      | 090          | 088     | 090           | 086     |
| 34       | 26          | M   | Closed transverse middle third of tibia  | Traffic         | Sliding tibial graft | 4                | 35   | 63  | 29               | 29      | 087          | 087     | 085           | 077     |
| 35       | 46          | F   | Closed oblique distal third of tibia     | Traffic         | Sliding tibial graft | 9                | 50   | 64  | 28               | 27      | 11           | 11      | 11            | 11      |
| 36       | 50          | F   | Closed oblique middle third of tibia     | Fall            | Screws               | 3                | 50   | 67  | 16               | 18      | 10           | 10      | 084           | 088     |
| 37       | 62          | F   | Closed comminute distal third of tibia   | Traffic         | Plaster              | 3                | 50   | 67  | 11               | 12      | 079          | 072     | 10            | 09      |
| 38       | 70          | M   | Closed comminute distal third of tibia   | Fall            | Screws               | 3                | 50   | 69  | 26               | 25      | 090          | 079     | 099           | 11      |



|    |    |   |   |         |             |    |    |     |    |    |     |     |     |     |
|----|----|---|---|---------|-------------|----|----|-----|----|----|-----|-----|-----|-----|
| 39 | 62 | F | Closed oblique<br>proximal third of tibia | Traffic | Lanes plate | 5  | 50 | 72  | 13 | 15 | 094 | 085 | 086 | 079 |
| 40 | 77 | F | Closed comminute<br>distal third of tibia | Traffic | Plaster     | 5  | 50 | 72  | 16 | 20 | 068 | 064 | 083 | 090 |
| 41 | 55 | F | Closed comminute<br>distal third of tibia | Fall    | Plaster     | 2½ | 50 | 81  | 13 | 13 | 068 | 070 | 076 | 068 |
| 42 | 70 | M | Open comminute<br>distal third of tibia   | Fall    | Plaster     | 8  | 50 | 81  | 21 | 22 | 14  | 18  | 11  | 10  |
| 43 | 68 | F | Closed oblique<br>distal third of tibia   | Fall    | Screws      | 3½ | 50 | 106 | 13 | 16 | 060 | 043 | 087 | 087 |

<sup>a</sup> All types of treatment included plaster

Fracture of the contralateral patella 4 months prior to the isotope investigation

Osteoma of the ipsilateral femoral shaft

TABLE II

*Delayed healing pseudarthrosis and/or osteitis*

| Case Fig       | Age (years) | Sex | Type and localization of fracture      | Cause of injury | Treatment (1)<br>(Figures show time in months after fracture)   | Clinical state at time of isotope investigation | Dose of $^{89}\text{Sr}$ ( $\mu\text{C}$ ) | Months between fracture and isotope injection | Activity ratios   |        |         |        |                |
|----------------|-------------|-----|--|-----------------|---|---|--|---|-------------------|--------|---------|--------|----------------|
|                |             |     |  |                 |   |   |  |   | Fracture/ control | 1 week | 2 weeks | 1 week | Thigh/ control |
| 1 17 31        | 57          | M   | Open comminute distal third of tibia   | Traffic         | Screws  | Delayed healing                                 | 50   | 6   | 92                | 136    | 13      | 19     | 16             |
| 2 32           | 19          | M   | Closed comminute middle third of tibia | Traffic         | Screws  | Delayed healing<br>Osteitis                     | 25   | 7   | 111               | 137    | 16      | 23     | 15             |
| 3 18 33        | 22          | M   | Open transverse middle third of tibia  | Traffic         | Egger's plate   | Delayed healing<br>Infection                    | 40   | 7   | 132               | 203    | 28      | 39     | 26             |
| 4 19 23 24 A-C | 38          | M   | Open comminute middle third of tibia   | Traffic         | Transfixation with Steinman pins  | Delayed healing                                 | 40   | 8½  | 74                | 80     | 26      | 36     | 21             |
| 5 35           | 47          | M   | Open transverse middle third of tibia  | Crush           | Egger's plate<br>Extr. of plate (5)<br>Sequestrectomy (9)   | Pseudarthrosis<br>Osteitis                      | 40   | 19  | 136               | 206    | 41      | 68     |                |
| 6 20 A-B 36    | 58          | M   | Open transverse middle third of tibia  | Traffic         | Egger's plate<br>Extr. of plate (2)<br>Skin transpl. (3)<br>Transfixation (15)<br>Osteotomy of fibula (20)<br>Amputation (26) | Pseudarthrosis                                  | 50   | 26  | 4.2               |        | 12      | 12     |                |
| 7 37           | 67          | M   | Open comminute distal third of tibia   | Crush           | Egger's plate<br>Extr. of plate and only bone graft (6)   | Pseudarthrosis                                  | 50   | 29  | 43                |        | 0.94    |        | 12             |
| 8 38           | 61          | M   | Open comminute distal third of tibia   | Crush           | Screws and wires<br>Extr. of wires and screws (5)<br>Curettage (14)   | Osteitis  | 50   | 42  | 34                | 42     | 13      | 15     | 11             |

All types of treatment included plaster

## **RADIOGRAPHS**

Fig 27 Radiograph of the fractured leg at the time of isotope investigation 5 1/2 months after injury of Case I-7

Fig 28 Radiograph of the fractured leg at the time of the isotope investigation 58 months after injury of Case I 33

Fig 29 A—C Radiographs of the fractured leg immediately after injury (A) after reposition of the fracture (B) and at the time of isotope investigation 15 months after injury (C) of Case I-23



Fig 27



Fig 28



Fig 29—A



Fig 29—B



Fig 29—C

Fig 30 Radiograph of the fractured leg at the time of isotope investigation 64 months after injury of Case I-3. A sliding bone graft has been taken proximal to the fracture

Fig 31 Radiograph of the fractured leg at the time of isotope investigation 6 months after injury of Case II-1. The tibial fracture showed delayed healing and the proximal fibular fracture non union

Fig 32 Radiographs of the fractured leg at the time of isotope investigation 7 months after injury of Case II-2. This case had osteitis in the fracture region

Fig 33 Radiograph of the fractured leg at the time of isotope investigation 7 months after injury of Case II-3. The fracture showed delayed healing and was later operated with only bone graft



Fig 30



Fig 31



Fig 32



Fig 33

Fig. 34 Radiograph of the fractured leg at the time of isotope investigation 8 ½ months after injury of Case II-4

Fig. 35 Radiograph of the fractured leg at the time of isotope investigation 19 months after injury of Case II-5. This case had a pseudarthrosis and osteitis

Fig. 36 Radiograph of the fractured leg at the time of isotope investigation 26 months after injury of Case II-6. This case had a pseudarthrosis of tibia and lower leg amputation was performed

Fig. 37 Radiograph of the fractured leg at the time of isotope investigation 29 months after injury of Case II-7. This case had a pseudarthrosis of tibia





Fig 34



Fig 35



Fig 36



Fig 37

Fig 38 Radiograph of the fractured leg at the time of isotope investigation 42 months after injury of Case II-8. The tibial fracture was healed but osteitis was present.

Fig 39 Radiograph of the knees of a patient who had a five week-old fracture of his right tibia (Case I-7). A slight loss of density was observed in the knee region of the fractured leg but the accretion rate in this knee did not differ from that in the other knee (see Fig 6 which shows the activity values in this patient).

The reproduction of this radiograph is hazardous but may be justified because separate interviews with five roentgenologists gave unequivocal evidence for loss of density in the right as compared to the left knee region.

Fig 40 Radiographs of the knees of a patient with a 6 1/2-month old fracture of his right tibia (Case I-10). An advanced loss of density was observed in the knee region of the fractured leg as compared to that of the intact leg. The externally recorded  $\text{Sr}^{90}$  activity in the right knee region at Day 14 was 2.6 times higher than that in the knee of the intact, left leg.



Fig 38



RIGHT  
(fractured leg)

LEFT  
(intact leg)

Fig 39



RIGHT  
(fractured leg)

LEFT  
(intact leg)

Fig 40







FROM THE ORTHOPAEDIC HOSPITAL OF THE INVALID FOUNDATION  
HELSINKI/HELSINGFORS HEAD A. LANGEŖSKIÖLD M.D AND  
THE DEPARTMENT OF PATHOLOGY UNIVERSITY OF HELSINKI/HELSINGFORS,  
MARIA HOSPITAL, HEAD E. SAXÉN M.D

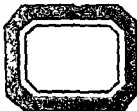
# THE REACTION TO MECHANICAL TRAUMA IN GROWING ARTICULAR CARTILAGE

*An experimental study on rabbits and a comparison  
of the results with the pathological anatomy of osteochondritis  
dissecans*

BY  
GUSTAV TALLQVIST

*Gustav Tallqvist*  
24/

MUNKSGAARD  
Copenhagen 1962







THE REACTION  
TO MECHANICAL TRAUMA IN GROWING  
ARTICULAR CARTILAGE



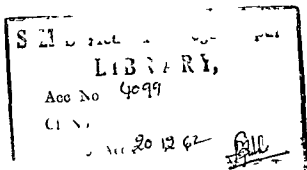
FROM THE ORTHOPAEDIC HOSPITAL OF THE INVALID FOUNDATION  
HELVINGHOLM HELSINGFORS HEAD A. LANGEN KJÖLD M.D. AND  
THE DEPARTMENT OF PATHOLOGY UNIVERSITY OF HELSINGFORS  
MARIA HOSPITAL, HEAD E. SANÉN M.D.

# THE REACTION TO MECHANICAL TRAUMA IN GROWING ARTICULAR CARTILAGE

*In experimental study on rabbits and a comparison  
of the results with the pathological anatomy of osteochondritis  
dissecans*

BY

GUSTAV TALLQVIST



Translated by IVA LAINEN



PRINTED IN FINLAND BY TILGMANN  
HELSINKI 196

*To my Wife*



# CONTENTS

|   | Page |
|---|------|
| Acknowledgements  | 9    |
| Introduction  | 11   |
| Survey of the literature  | 13   |
| <i>The embryology of cartilage</i>  | 13   |
| Vascularization and ossification of the epiphyse  | 13   |
| The skeletal development of the rabbit as compared with that of man   | 14   |
| Anatomy of the articular cartilage  | 15   |
| Nutrition of the articular cartilage  | 15   |
| Regressive changes in cartilage   | 15   |
| The regenerating capacity of cartilage  | 16   |
| The reaction of cartilage to tranplantation   | 17   |
| Experimentally created joint mice   | 17   |
| Certain other experiments performed on articular cartilage  | 18   |
| Certain features of the pathological anatomy of osteochondritis dissecans                                       | 18   |
| Material and methods  | 1    |
| Results   | 4    |
| Series A large fragment   | 4    |
| Summary of the results in series A  | 44   |
| Series B small fragment   | 48   |
| Summary of the results in series B  | 60   |
| Series C hinge of ligament tissue   | 64   |
| Summary of the results in series C  | 7    |
| Series D loose fragment   | 74   |
| Summary of the results in series D  | 8    |
| Series E intracartilaginous cleavage  | 84   |
| Summary of the results in series E  | 9    |
| Discussion and conclusions  | 94   |
| The present results in the light of the relevant literature   | 94   |
| The present results as compared with the pathological anatomy of osteochondritis dissecans                      | 100  |
| The present results as compared with the data on experimentally induced imitations of osteochondritis dissecans | 103  |
| Summary   | 105  |
| References  | 108  |





## ACKNOWLEDGEMENTS

The theme of this study was suggested to me by A. LANGEN KJELD M.D. Surgeon in Chief of the Orthopaedic Hospital of the Invalid Foundation, Helsinki, for his constant encouragement for most valuable criticism and for what he has taught me about scientific thinking, I wish to express my sincere gratitude.

I am also indebted to L. SYRÉN M.D. Professor of Pathology for criticizing the histological part of the study.

I owe particular gratitude to P. JORTELIUS M.D. who generously devoted much time to the checking of the histological sections. For assistance with the experiments my thanks are due to Miss I. MIKKOLA. Furthermore, I wish to thank Miss R. KOSKELA who prepared the histological sections and Mr. I. KORHONEN who took great pains in the production of the micrographs.

This investigation was aided by grant from the Sigrid Juselius Foundation and the Finska Läkarellskapet (Finnish Society of Physicians).

Helsinki November 1961

*Gustaf Tallqvist*



## INTRODUCTION

In 1933 A. LANGE-SKJOLD published a paper called «Can Osteochondritis Dissecans Arise as a Sequel of Cartilage Fracture in Early Childhood?» It was an experimental study based on a series of 12 rabbits. By surgical intervention at an early age a mechanical lesion of the cartilage of the medial femoral condyle was induced and some 100 days later the radiological and histological findings resembled to such a degree those of osteochondritis dissecans that the hypothesis cited above seemed to be justified.

The experimental lesion of the knee joint was described as follows. «With a small sharp scoop a fragment of cartilage 1 by 2 by 2 mm in size was gouged from the medial condyle so that it remained attached to the femur only by a narrow bridge of synovial tissue near the edge of the intercondylar fossa. This fragment consisted of pure cartilage and the concavity produced in the joint cartilage did not reach the epiphyseal bony nucleus». The results were described in the following way. «Ten animals were killed after a period varying between 33 and 116 days after operation. In eight of these a typical joint mouse containing a bony nucleus surrounded by cartilaginous tissue was found — — — The diameter of the joint mice was about twice that of the original cartilage fragments separated at operation — — — Seven joint mice produced experimentally and containing bone were examined histologically — — — the bony nucleus in each of them was entirely surrounded by a cartilaginous layer. The layer facing the joint cavity was considerably thicker than that facing the concavity. The structure of the former layer was fairly regular and similar to normal joint cartilage, the latter being irregular and consisting in part of fibrocartilage. The concavity was lined with a layer of irregular connective tissue or fibrocartilage. The bone tissue and the marrow of two of the joint mice seemed necrotic while the others contained living bone and marrow. There were abundant rests of cartilaginous tissue in the bony nuclei. Furthermore LANGE-SKJOLD found that «the fragment grows at about the same rate as the condyle and it maintains its shape and function as a part of the condyle».



## SURVEY OF THE LITERATURE

### The embryology of cartilage

When cartilage develops from mesenchymal tissue the intercellular substance disappears and the connective cellular process become shorter. The intercellular substance diminishes and finally disappears so that the cells form a syncytium. In this the cytoplasm adjacent to the nuclei change into cartilage cells while the remaining mass of the syncytium develop into the matrix or ground substance of the cartilage. By growth of the ground substance the cartilage cells are pushed farther and farther apart and between the cells collagen fibrils are formed in the matrix. The latter is saturated with chondrin and thus assume a uniform structure and appearance. The cartilage cells divide and form small groups of cells. Between the cells in the groups too ground substance develops so that the cells are pushed apart. This mode of growth is called interstitial growth. When the cartilage is surrounded by connective tissue it also grows by addition of new layers of tissue which is called appositional growth (BROMAN & HANCOCK 1944).

### Vascularization and ossification of the epiphyses

With regard to the vascularization and ossification of the foetal epiphyses there is no essential developmental difference in the rabbit, rat, guinea pig, and man (STUMP 1924). Initially the foetal epiphyses consist only of cartilage. During the second third of foetal life they are invaded by primitive connective tissue (STUMP 1924) which together with blood vessels penetrate into the cartilage through canals before ossification begins (WATT 1928, HAINES 1933, HUNNELL 1934, RICE 1935). During the first 2 days after birth the cartilage canals are still vascular but contain, on the 3rd day, how that the canals are occupied by proliferating connective tissue and no erythrocytes can be seen (RICE 1935). RICE believed that the ossification of the cartilage is due to the invasion of new vessels from the perichondrium

The relevant literature is of little help in attempting to account for the phenomenon described above. DAVIS & LAMARCA (1957) took a piece of fibrous cartilage from fracture callus and implanted it into the anterior chamber of the eye of rat where the fragment ossified. SICCA (1961) and LACROIX (1959) reported similar results with articular cartilage implanted into the eye under the renal capsule or into the peritoneal cavity.

On the other hand, FANDELIS (1957) stated that cartilage may live, grow and calcify (but not ossify) when completely detached from the bone provided it has free access to the synovial fluids.

There is no evidence of cartilage ossifying without preceding vascularization. FRELTA (1958) suggested that the osteoblasts are developmental forms of the endothelial cells of the capillaries.

An attempt therefore at elucidating the mechanism of the phenomenon described by LANCENSKIOLD necessitated a study of the vascularization of the piece of cartilage. The fragment had been detached except for a narrow bridge of synovial tissue. Did vascularization of the loose body take place via this piece of tissue? Were there any other routes by which the invasion of capillaries was possible? To what extent was the reference to osteochondritis dissecans made by LANCENSKIOLD justified? Did he only demonstrate a close resemblance between two phenomena which were independent of each other? Or, it possible to contribute to the clarification of the genesis of osteochondritis dissecans by studying in detail the phenomenon described by LANCENSKIOLD?

The purpose of the present study was to throw further light on the mechanism and conditions of the phenomenon described by LANCENSKIOLD and if possible to answer the questions posed above.

## SURVEY OF THE LITERATURE

### The embryology of cartilage

When cartilage develops from mesenchymal tissue the elongated mesenchymal cells of the latter grow and the connecting cellular processes become shorter. The intercellular substance diminishes and finally disappears so that the cells form a syncytium. In this the cytoplasm adjacent to the nuclei changes into cartilage cells while the remaining mass of the syncytium develops into the matrix or ground substance of the cartilage. By growth of the ground substance the cartilage cells are pushed farther and farther apart and between the cells collagen fibrils are formed in the matrix. The latter is saturated with chondrin and thus assumes a uniform structure and appearance. The cartilage cells divide and form small groups of cells. Between the cells in the groups too ground substance develops so that the cells are pushed apart. This mode of growth is called intussusception. When the cartilage is surrounded by connective tissue it also grows by addition of new layers of tissue which is called appositional growth (BROMAN & HANSSON 1941).

### Vascularization and ossification of the epiphyses

With regard to the vascularization and ossification of the foetal epiphyses there is no essential developmental difference in the rabbit, rat, guinea pig and man (STUMP 1924). Initially the foetal epiphyses consist solely of cartilage. During the second third of foetal life they are invaded by primitive connective tissue (STUMP 1924) which together with blood vessels penetrates into the cartilage through canals before ossification begins (WATT 1928; HAINES 1933; HURRELL 1934; RICE 1935). During the first 2 days after birth the cartilage canals are still vascular but sections on the 3rd day show that the canals are occupied by proliferating connective tissue and no erythrocytes can be seen (RICE 1935). RICE believed that the ossification of the cartilage is due to the invasion of new vessels from the perichondrium

when the vascularity of the old cartilage canals has decreased and the nutrition of the surrounding cartilage has been disturbed.

Endochondral ossification is similarly described by most writers and the course is the same in the diaphyses and epiphyses of the long bones as in the short bones. The ossification of the epiphyses of the long bones develops in all respects as though it were an independent bone and the process is exactly similar to that found in the short irregular bones such as the tarsals and carpals (WATT 1928). Ossification is preceded by typical structural changes in the centre of the cartilaginous epiphysis. The cartilage cells increase in size and vacuoles develop in their peripheral cytoplasm. The nuclei swell and lose some of their chromatin. The matrix surrounding these cells decreases in volume. According to many authors blood capillaries now penetrate from the periphery of the epiphysis (BROMAN & HALLGREN 1941; BENNINGHOFF 1949; RICE 1955; HAM 1957; MAXIMOW & BLOOM 1958; FRUETA 1958). The matrix surrounding the hypertrophic cartilage cells calcifies and the cells die. The resulting cavities are invaded by capillaries, primitive bone is deposited at the walls of the cavities and by the course of development spongy bone is formed. Thus a bony nucleus develops which grows concentrically.

In the new born child the cartilage of the distal femoral epiphysis is supplied by vessels some of which penetrate along the margins of the condyles and anastomose with each other in the cartilage of the condyles. The centre of the epiphysis is supplied by vessels running near the site of the future epiphyseal plate. This vascular pattern is maintained in adult life (LOCK 1960). In the adult rabbit the corresponding vessels are similarly arranged (BROOKES & HARRISON 1957). It may be assumed that the pattern is the same in the new born rabbit (MORGAN 1960).

### **The skeletal development of the rabbit as compared with that of man**

HEIKIL (1960) stated that both man and the rabbit have at birth or shortly after a radiologically demonstrable nucleus in the distal femoral epiphysis and the proximal tibial epiphysis but in no other epiphysis. It may therefore be concluded that they are both born at almost the same skeletal ages. Hence the rabbit seems to be a suitable laboratory animal for experiments involving the distal femoral epiphysis. The whole growth period of the rabbit to the time of final ossification of the epiphyseal plates covers



some 220 days. After the age of 100 days however growth is slight (HELIEL 1960). For practical purposes therefore the first 100 days of life may be regarded as covering the growth period of the rabbit.

### **Anatomy of the articular cartilage**

In the adult articular cartilage has a typical structure the collagen strands in the matrix forming arches placed at right angles to the joint surface. The cartilage cells too arrange themselves in incomplete rows following the same pattern. The system of collagen fibrils in articular cartilage is a direct continuation of the collagen fibrils in the periosteum. Nerves and blood vessel are lacking in adult articular cartilage (BRUNNENHOFF 1949 HAN 1957 MAXIMOW & BLOOM 1957).

### **Nutrition of the articular cartilage**

The articular cartilage is believed to be supplied in part by the subchondral blood vessels in part by the synovial fluid (STRANCEWAYS 1920 MOORE 1948 INCELMARK & SAAF 1948 HIRSCH 1951 BAILEY & SELLI 1959). Direct contact between the subchondral marrow cavities and the basal parts of the articular cartilage was demonstrated by HOLMDAHL & INCELMARK (1951). Since the points of contact were more numerous where the articular cartilage is not normally exposed to great pressure and less numerous where greater pressure is exerted the writers concluded that compression may be a factor of significance in the transportation of fluid to and from the articular cartilage. Similar views were expressed by GIBSON (1955).

### **Regressive changes in cartilage**

The following four phenomena are generally accepted as signs of decreased vitality in cartilage: a) vacuolization and accompanying hypertrophy of the cytoplasm in the cell; b) decreased or suspended stainability of the nuclei of the cell which initially may be of normal size and shape but later become pyknotic; c) calcium deposits in the matrix; d) formation of parallel fibres in the ground substance which have nothing in common with the collagenous fibres of the cartilage (asbestos transformation). Simultaneously

when the vascularity of the old cartilage canals has decreased and the nutrition of the surrounding cartilage has been disturbed.

*Endochondral ossification* is similarly described by most writers and the course is the same in the diaphyses and epiphyses of the long bones as in the short bones. The ossification of the epiphyses of the long bone develops in all respects as though it were an independent bone and the process is exactly similar to that found in the short irregular bones such as the tri-rals and carpals (WERT 1928). Ossification is preceded by typical structural changes in the centre of the cartilaginous epiphysis. The cartilage cell increases in size and vacuoles develop in their peripheral cytoplasm. The nuclei swell and lose some of their chromatin. The matrix surrounding these cells decreases in volume. According to many authors blood capillaries now penetrate from the periphery of the epiphysis (BROMAN & HANSSON 1911; BLANKENHOFF 1949; RICE 1955; HAY 1957; MAXIMOW & BLOOM 1958; FULTON 1958). The matrix surrounding the hypertrophic cartilage cell calcifies and the cells die. The resulting cavities are invaded by capillaries. Primitive bone is deposited at the walls of the cavities and by the course of development spongy bone is formed. Thus a bony nucleus develops which grows concentrically.

In the new born child the cartilage of the distal femoral epiphysis is supplied by vessels some of which penetrate along the margins of the condyles and anastomose with each other in the cartilage of the condyles. The centre of the epiphysis is supplied by vessels running near the site of the future epiphyseal plate. This vascular pattern is maintained in adult life (CROCK 1960). In the adult rabbit the corresponding vessels are similarly arranged (BROOKES & HARRISON 1957). It may be assumed that the pattern is the same in the new born rabbit (MORRIS 1960).

### **The skeletal development of the rabbit as compared with that of man**

HEIKEL (1960) stated that both man and the rabbit have at birth or shortly after a radiologically demonstrable nucleus in the distal femoral epiphysis and the proximal tibial epiphysis but in no other epiphysis. It may therefore be concluded that they are both born at almost the same skeletal age. Hence the rabbit seems to be a suitable laboratory animal for experiments involving the distal femoral epiphysis. The whole growth period of the rabbit to the time of final ossification of the epiphyses and plates covers

tion. A similar course was observed when an infraction of the subchondral bone was created and the articular cartilage was left intact (NAGURA & KOSICE 1938 BURCKHARDT 1948)

### The reaction of cartilage to transplantation

It is a common observation that cartilage when transplanted maintains its vitality longer than other tissues (SEGGER 1904 ANHAUSEN 1912 RAO 1951 HAM 1957 BALLANTYNE & CONVERSE 1958 BAXTER GOLDSTEIN & McMILLAN 1958 CRAIGMYLE 1958 DE PALMA 1960). It has been reported that mitoses do not occur but that the cells nevertheless remain alive (HAM 1957 BALLANTYNE & CONVERSE 1958). The criteria generally accepted for vitality of cartilage are normal stainability, shape and size of the cartilage cells and their nuclei and normal stainability and structure of the matrix. The capacity of cartilage for surviving transplantation has been attributed to its slow metabolism (BYWATERS 1937 HAM 1957). It has been observed that cartilage which normally does not ossify in its original site does not ossify after transplantation either. By contrast foetal joint cartilage or cartilage taken from fracture callus ossifies when implanted into the eye or under the renal capsule in laboratory animals (DANIS & LA BARRE 1957 LACROIX 1959). GIES (1882) implanted embryonic cartilage into the knee joint of dog and observed in one case that the fragment was surrounded by connective tissue peripherally and degenerated centrally, in another case that the fragment was completely resorbed. SLOCUM (1904) excised a piece of articular cartilage from the femoral condyle of young rabbits (weighing under 2 kg) and implanted it into the peritoneal cavity. He observed that the graft was converted into bone tissue. RAO (1954) took a piece of cartilage from the xiphisternum of 6 month old rats and implanted it into the peritoneal cavity. In one case out of 48 the graft was in part converted into osteoid tissue.

### Experimentally created joint mice

A large number of studies have been reported involving the experimental production of joint mice. The results have been largely similar.

When a piece of cartilage is mechanically detached from the joint surface without injuring the subchondral bone the fragment is either resorbed or reunites with the joint capsule or it is later found as a loose body in the joint cavity (GIES 1882 HILDEBRAND 1896 STRANGEWAYS 1920

BENNET BAUER & MADDOCK (1932) Initially the piece of cartilage seems to maintain its vitality but later it shows degenerative changes. Although the fragment when it unites with the joint capsule is surrounded by vascular connective tissue it is not invaded by blood vessel nor does it ossify (BENNET BAUER & MADDOCK 1932).

When a piece of cartilage together with a piece of underlying bone tissue is mechanically separated from the joint surface the fragment unites with the site from which it has been detached or it unites with the joint capsule or with that part of the joint which is not lined with cartilage (BARTH 1898 CORNIE & COUDRAY 1900 RIMANN 1900 ANHAUSEN 1923 SCHMIDT 1931). In certain cases the fragment is completely reabsorbed. The investigations cited above were all performed on adult or nearly adult animal.

### Certain other experiments performed on articular cartilage

According to NACURA (1937) and BURCKHARDT (1948) a growing animal divided differs from an adult with regard to the reaction of the articular cartilage and the subchondral bone to incision. These writers used 15 to 19 day old rabbits. Their experimental techniques and results were broadly speaking similar. After incision of the articular cartilage and the underlying bone NACURA reported complete healing of the cartilage. The cleavage in the subchondral spongy bone was filled with a cartilage like tissue which showed a tendency towards enchondral ossification. This process was strikingly slow and several months after operation changes in the surroundings of the lesion were still prominent.

RIMANN (1930) subjected the distal femoral epiphysis in adult dog to repeated blunt trauma. During an observation time of 18 days the injured site exhibited an area of necrotic bone and cartilage limited by a streak of vascular loose tissue. Proximal to this streak osteoblasts were seen in large numbers and distally there was extensive reorption of the bone tissue. Radiologically the findings resembled osteochondritis dissecans.

### Certain features of the pathological anatomy of osteochondritis dissecans

*The macroscopic finding.* On gross inspection of the joint surface the findings may be completely negative (KROHN 1928) or there may be an area of the joint surface differing from the surrounding tissue in that it exhibit

a paler yellow white colour or may yield elastically to pressure (COLVIN 1920 LEHMANN 1923) or it may be relatively rigid (KROH 1928 KING 1932). The affected area may be bounded by a hollow core in the joint surface or by a cleavage which more or less completely surrounds and separates it from the surroundings and from the underlying tissue (COLVIN 1920 LEHMANN 1923 KROH 1928). In the latter case a fragment of cartilage or of cartilage and bone is found in a concavity of corresponding size. The fragment may adhere to the concavity by a bridge or a hinge of intact articular cartilage connective tissue or bone tissue (LEHMANN 1923 KING 1932 DE PALMA 1955). The fragment may alternatively be found as a loose body in the joint in which case fragment and concavity seldom correspond to each other with regard to shape. Growth of the separate fragment *in situ* has been observed radiologically by certain writers (FRIBERG 1923 SCHELLER 1960 JACROIX FIEVEZ & CAMBIER 1961 LANCENSKJOLD unpublished observation) while others have found that the fragment grew when it was a loose body (PHEMISTER 1924 DE PALMA 1955).

*Microscopic findings.* A concavity in the subchondral zone of the bone is typical of osteochondritis dissecans. As a rule a piece of bone is found in the concavity separated from the epiphyseal bone by connective tissue or by cartilage. The latter may be fibrous (RIEBIN 1901) or hyaline (LEHMANN 1923) and it may be completely or partially divided by a cleavage into a layer lining the concavity and another lining the corresponding surface of the fragment. In certain cases the cleavage reaches a point just under the joint surface (LEHMANN 1923); in others it includes the joint surface. As a rule fibrous cartilage is found on both sides of the cleavage (FAIRBANK 1933) but hyaline cartilage (NEUMAN & SUTER 1918 LEHMANN 1923 BIANCHI GOIDANACH & ZANASI 1955) vascular connective tissue (KIRSCHNER 1901 COLVIN 1920 KAPPEL 1920 STORÉN 1934 young cartilage (LACROIX 1911) and islands of hyaline cartilage in fibrous cartilage (LEHMANN 1923) have also been observed. According to the majority of writers the bone tissue in the separate fragment is necrotic particularly when the fragment is a loose body in the joint (MARTENS 1893 BOERNER 1903 KIRSCHNER 1901 LEB 1924 LEHMANN 1925 ROMPOLD 1936 BIANCHI GOIDANACH & ZANASI 1955 SMITH 1960). In a fragment retained in its concavity many investigators have observed living bone or living bone together with necrotic bone (BARTH 1898 MARTEN 1899 BOERNER 1903 KAPPEL 1917 FISCHER 1921 LEHMANN 1923 KING 1932 FAIRBANK 1933 LINDEMANN 1937 JACROIX FIEVEZ & CAMBIER 1961). Furthermore the centre of the fragment has been reported to exhibit osteoid tissue (KROH

BISSNET BAUER & MADDOCK 1932). Initially the piece of cartilage seems to maintain its vitality but later it shows degenerative changes. Although the fragment when it unites with the joint capsule is surrounded by vascular connective tissue it is not invaded by blood vessels nor does it ossify (BISSNET BAUER & MADDOCK 1932).

When a piece of cartilage together with a piece of underlying bone is surgically separated from the joint surface the fragment unites with the site from which it has been detached or it unites with the joint capsule or with that part of the joint which is not lined with cartilage (BARTU 1898; CORNILL & COUDRAY 1900; RIMANN 1900; ANHANGSEN 1923; SCHMIDT 1924). In certain cases the fragment is completely reabsorbed. The investigations cited above were all performed on adult or nearly adult animal.

### **Certain other experiments performed on articular cartilage**

According to NACHT (1937) and BURCHARDT (1948) a growing animal differs from an adult with regard to the reaction of the articular cartilage and the subchondral bone to incision. The writers used 15 to 19 day old rabbit. Their experimental technique and results were broadly speaking similar. After incision of the articular cartilage and the underlying bone NACHT reported complete healing of the cartilage. The cleavage in the subchondral spongy bone was filled with a cartilage like tissue which showed a tendency towards enchondral ossification. This process was strikingly slow and several months after operation changes in the surroundings of the lesion were still going on.

RIMANN (1900) subjected the distal femoral epiphysis in adult dog to repeated blunt trauma. During an observation time of 48 days the injured site exhibited an area of necrotic bone and cartilage limited by a streak of vascular loose tissue. Proximal to this streak osteoblasts were seen in large number and distally there was extensive reabsorption of the bone tissue. Histologically the findings resembled osteochondritis dissecans.

### **Certain features of the pathological anatomy of osteochondritis dissecans**

*The macroscopic finding.* On gross inspection of the joint surface the findings may be completely negative (KRON 1928) or there may be an area of the joint surface differing from the surrounding tissue in that it exhibits

## MATERIAL AND METHODS

The material consisted of a total of 111 rabbits aged one to 12 days divided into five series including 16—40 animals each. In each series the animals were operated upon in a way which was typical of the series. The animals were sacrificed one at a time at various intervals up to an age of 102 to 200 days.

*General operative technique.* Under aseptic conditions arthrotomy of the right knee joint was performed under local anaesthesia (0.25 per cent Xylocain) while the left knee was left intact as control. A longitudinal ventral skin incision was made and the joint was opened by parapatellar approach. The patella and the quadriceps tendon were dislocated in the lateral direction and the joint surface of the medial condyle was exposed by flexion of the joint. The operation was performed on the articular cartilage of the medial condyle and the resulting lesion was carefully measured. Operation and measurement were performed using a binocular preparation microscope and magnification 4—25 $\times$  which enabled measurement to the nearest 0.1 mm. Then the joint was closed in layers with catgut. No dressing was applied. Infection developed in two cases which were omitted from the report.

*Method of investigation.* Post mortem the knee joints were cautiously opened. All noticeable deviations from the normal control side were *measured* and *photographed* using the above mentioned preparation microscope. In addition both femora were freed from all soft parts and *roentgenograms* were taken in at least two planes at right angles to each other. After fixation in neutral formalin and decalcination in EDTA (ethylenediaminetetraacetic acid) the preparations were embedded in paraffin and sectioned at 10  $\mu$  in either the frontal or the sagittal plane. The sections were stained with haematoxylin and van Gieson. Sectioning was performed in an incomplete series throughout the preparation which rendered *histological examination* of all parts of the specimen possible.

The following operations were performed in the different series.

*Series 1: large fragments.* With a small sharp scoop a fragment of cartilage

1928 FAIRBANK 1933) living bone marrow and erythrocytes (LEHMANN 1921). In cases showing a direct connection between the fragment and the bone tissue of the concavity osteoblasts and osteoid tissue have been observed in the proximity of this bridge while the bony nucleus has otherwise been necrotic. In addition islands of hyaline cartilage in the bony nucleus of the fragment have been observed (LEHMANN 1921). Many writers have observed that the bone trabeculae in the fragment are thicker and the marrow cavities narrower than in the surrounding subchondral bone (LEHMANN 1921, BIANCHI, GOIDANACHI & ZANASI 1911, and others). The part of the fragment facing the joint usually consists of hyaline cartilage with a normal structure and living cells (KIRSCHEN 1909, WEIL 1921, LEHMANN 1921, ROMBOLD 1936). Only occasionally does it exhibit regressive changes (PUMMSTEIN 1924). A common observation is that this hyaline layer of cartilage is not equally thick throughout but increases in thickness at the periphery of the fragment thus forming a frame around the underlying bony nucleus (LEW 1924, LEHMANN 1921, LACROIX 1911, BIANCHI, GOIDANACHI & ZANASI 1911). LACROIX (1911) showed that the cartilage cells in this marginal area are arranged in parallel with the cleavage surrounding the fragment.

In cases where the fragment is completely detached from the concavity the margins of the latter are rounded and in later stages the concavity itself is filled with fibrous cartilage which is pushed deeper down (KROHN 1928, FAIRBANK 1933).



like with the shape of a crescent was separated from its surrounding the most superficial layer of the joint cartilage excepted (Figs 93 and 94 page 8). The piece was made about 1.0 mm thick and included the whole breadth of the condyle in the plane of the joint surface.

A total of 18 animals were operated upon at an age of 6 to 8 days and sacrificed at different interval up to an age of 150 day.

## RESULTS

In the following account of the results cases are described which were typical of the postoperative development in the series in question.

### *Series A «large fragment»*

#### Stage 1 (series A)

This is the situation immediately after operation 1 (for details see page 21 and the opposite page Figs 1 and 2).



Fig. 1

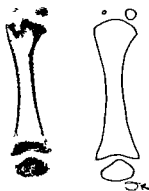


Fig. 2

### Stage 2 (series A)

This time is represented by an animal sacrificed 7 days after operation.

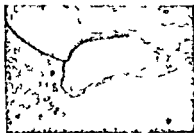
*Microscopic findings:* The fragment lies in a corresponding concavity and the ossification of the epiphysis has proceeded almost to the bottom of this (Fig. 3). Figure 5 shows that the cleavage between fragment and concavity is filled with loose young granulation tissue with fibrin in the meshes and newly formed capillaries. At the bottom of the concavity a communication with the vessel of the ossified zone is seen (Fig. 4). At the medial margin of the fragment the cleavage is bridged by proliferating vascular connective tissue originating in the perichondrium and penetrating between the fragment and the concavity (Fig. 6).



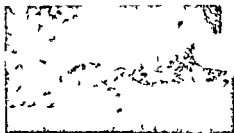
Г. 3



Г. 4



Г. 5



Г. 6

### Stage 3 (series A)

This stage is represented by an animal sacrificed 13 days after operation.

*Microscopic findings* The fragment lies in a corresponding concavity. The ossification of the epiphysis has proceeded so far that in the zone of ossification at the site of the lesion a shallow concavity has developed in contrast to the convexity seen in the intact lateral condyle (Fig. 7). The cleavage between fragment and concavity contains granulation tissue rich in cells which particularly in the lateral part of the cleavage resembles primitive cartilage (Fig. 8). The granulation tissue is highly vascular and the vessels anastomose with the vessels in the perichondrium of the medial side, the vessels in the intercondylar fossa (Fig. 8) and the vessels in the marrow cavities of the ossified zone. From the granulation tissue in the lateral part of the cleavage a small protrusion surrounded by osseous tissue penetrates into the cartilaginous mass of the fragment (Fig. 8). Another section shows a transverse vessel penetrating from the intercondylar fossa into the lateral portion of the fragment. Apparently this vessel is not surrounded by osseous tissue (Fig. 9). The medial portion of the cleavage is bridged by vascular connective tissue originating in the perichondrium.

The roentgenogram shows a shallow concavity in the outline of the subchondral bone of the operated medial condyle (Fig. 10).



Fig. 7



Fig. 8

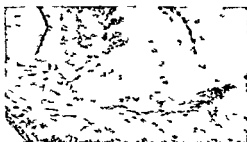


Fig. 9

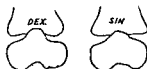


Fig. 10

### Stage 4 (series A)

The stage is represented by an animal sacrificed 16 days after operation.

*Microscopic findings* The fragment lies in its concavity (Fig. 11). The concavity is separated from the subchondral zone of ossification by a thin layer of cartilage (Fig. 12). The cleavage between fragment and concavity is variable, and the innermost part of it is filled with vascular granulation tissue rich in cells. The medial part of the cleavage is bridged by vascular connective tissue originating in the perichondrium. Within the cartilaginous mass of the fragment there is a small centre of ossification near the bottom of the concavity (Fig. 12). This ossific centre shows a tend to be and blood vessels which communicate with the vessels of the epiphyseal spongy bone via the vascular granulation tissue in the lateral portion of the concavity. Where the vessels penetrate the cartilage minute ossific trabeculae are seen.

The *roentgenogram* shows a concavity in the outline of the subchondral bone. At the bottom of the concavity there is a separate density in the cartilage shadow (Fig. 13).





Fig. 11



Fig. 1



Fig. 13

### Stage 5 (series A)

This stage is represented by an animal sacrificed 13 days after operation.

*Microscopic findings.* The fragment lies in its concavity (Fig. 14). Most of the cleavage between fragment and concavity is clearly visible and contains both vascular proliferation tissue rich in cells and hyalinized connective tissue (Fig. 15). Medially the cleavage is bridged by vascular connective tissue originating in the perichondrium. The ossification of the epiphysis has proceeded so far that the fragment is in part ossified (Fig. 16). Within the cartilage of the fragment there is a separate centre of ossification with marrow cavities filled with blood vessels and connective tissue (Fig. 17). This separate ossific centre is supplied by blood vessels coming from the proximal bone of the epiphysis via the vascular proliferation tissue in the cleavage (Fig. 18). In the cartilage adjacent to this vascular communication osteoid tissue is seen.

The roentgenogram shows a concavity in the outline of the subchondral bone. At the bottom of the concavity a separate bone shadow is seen (Fig. 19).



Fig 14



Fig 15

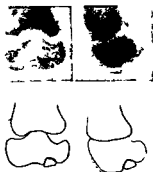


Fig 16

### Stage 6 (series A)

This stage is represented by an animal sacrificed 22 days after operation.

*Microscopic findings* The fragment lies in its concavity. Ossification of the epiphysis has proceeded to near the concavity and the spongy bone is separated from the latter by a thin layer of hyaline cartilage. In the zone of ossification a corresponding concavity is thus formed (Fig. 17). In the fragment a separate bony nucleus is seen which has a biconvex shape in cross section in the frontal plane. The centre of ossification is covered by hyaline cartilage which is thick in the aspect facing the joint and thin in the aspect facing the concavity (Fig. 17 and 18). Most of the cleavage between fragment and concavity is visible. Medially it is bridged by dense connective tissue originating in the perichondrium. Laterally it is filled with primitive cartilage which in some parts shows a gradual transition into older cartilage while in other parts there is a clear line of demarcation (Fig. 18). The vessels in the separate centre of ossification in the fragment communicate with the vessels in the intercondylar fossa. This communication runs through the cartilage of the hinge (Fig. 18). At the same site there is also a vascular connection with the epiphyseal spongy bone.

The *roentgenogram* shows a concavity in the outline of the subchondral bone of the medial condyle. In the concavity a separate bone shadow is seen (Fig. 19).



Fig 17

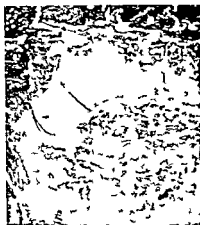


Fig 18

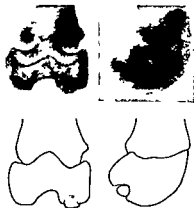


Fig 19

### Stage 3 (series A)

This stage is represented by an animal sacrificed 23 days after operation.

*Microscopic findings* The joint cartilage of the medial condyle is thicker than that of the lateral condyle (Fig. 24). The bone of the medial condyle is penetrated by a transverse curved streak of cartilage and osteoid tissue (Fig. 25). In the continuation of this streak near the aspect of the condyle facing the intercondylar fossa there is an island of hyaline cartilage within the spongy bone (Fig. 25 arrow). Medially the streak consists of a protruding wedge of two layers of hyaline cartilage with highly vascular granulation tissue between them (Fig. 25).

The *roentgenogram* shows a crescent shaped indistinct line of demarcation crossing the bone of the medial condyle (Fig. 26).

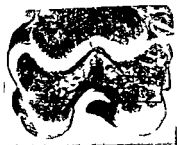


Fig. 94



Fig. 95

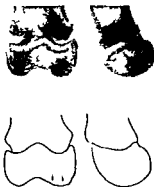


Fig. 96

### Stage 9 (series A)

This stage is represented by an animal sacrificed 55 days after operation.

*Microscopic findings* A section made in the sagittal plane shows at the site of the former cleavage between fragment and concavity a discontinuous streak of hyaline cartilage penetrating into the spongy bone (Fig. 27). At the site of the former cleavage in the plane of the joint surface there is a portion where the cartilage cells are irregularly arranged, compared with the structure of the adjacent cartilage (Figs. 28, 29 and 30). In the plane of the joint surface the border of the former fragment is in some parts even in others it is characterized by a shallow indentation (Fig. 27). The streaks of hyaline cartilage which penetrate into the spongy bone show signs of enchondral ossification (Fig. 28).

The *roentgenogram* shows an incomplete line of demarcation between a concavity in the outline of the subchondral bone and a bone shadow filling this concavity (Fig. 31).



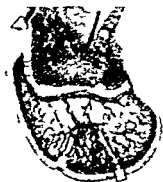


Fig. 28



Fig. 29



Fig. 30



Fig. 31



Fig. 32

### Stage 10 (series A)

This stage is represented by an animal sacrificed 91 days after operation.

*Microscopic findings* A section made in the sagittal plane shows that the structure of the subchondral bone is normal in the main. At one site in the joint cartilage there is at right angles to the joint surface a cleavage which penetrates for some distance into the subchondral spongy bone. The cleavage is lined with layers of hyaline cartilage which become thinner towards the bottom of the cleavage. In these layers the cells are mainly arranged parallel with the cleavage and perpendicularly to the joint surface (Figs 32 and 31). More ventrally at the site of the former cleavage there is joint cartilage with cells irregularly arranged as compared with the adjacent cartilage in which the cells are arranged in clusters (Figs 33, 35 and 36). The structure of the underlying bone does not deviate from that of the control side.

The *roentgenogram* shows that the medial condyle is somewhat deformed. In the lateral view an indistinct break penetrates into the subchondral bone (Fig. 37).



Fig 32



Fig 33



Fig 34



Fig 35



Fig 36



Fig 37

### Summary of the results in series A

In all animals in this series the course of development was that described in the foregoing as stages 1—10 (see Plates I—III). The figures in the plates (denoted a) are contour drawings of micrographs and roentgenograms with corresponding numbers shown in connection with the description of the various stages.

When the cartilaginous fragment was partially detached a concavity was formed in the subchondral zone of ossification (stages 1—3). In the fragment a separate centre of ossification developed which grew and finally fused with the rest of the condyle (stages 4—10).

The fragment grew as a part of the condyle and at the same rate as this. The bony nucleus of the fragment was supplied by vessels coming from the spongy bone of the remaining epiphysis via highly vascular granulation tissue in the cleavage between the fragment and the concavity and by vessels originating in the ligament tissue in the intercondylar fossa.

In the cleavage between the fragment and the concavity the following structures succeeded each other: highly vascular granulation tissue, hyalinized connective tissue, primitive cartilage and hyaline cartilage. Gradually the cleavage disappeared and was replaced by a solid wall of hyaline cartilage which was divided into fragments by enchondral ossification and which finally ossified completely. In the plane of the joint surface the cleavage between the fragment and the concavity persisted in some places while in others the edges of the cleavage united by the formation of hyaline cartilage of irregular structure.

## PLATE I

Stage 1



Fig 1 a

Fig 2 a

Stage 2



Fig 3 a

Stage 3



Fig 4 a

Fig 10 a

Stage 4

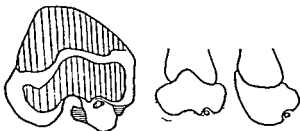


Fig 11 a

Fig 13 a

Stage d

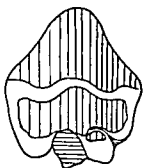


Fig 14 a



Fig 16 a

Stage e



Fig 17 a

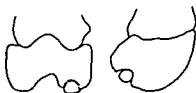


Fig 19 a

Stage f

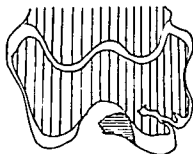


Fig 20 a



Fig 23 a

Stage g

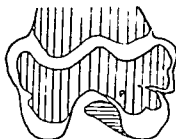


Fig 24 a



Fig 25 a

## PLATE III

Stage 9

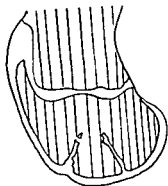


Fig 27 a



Fig 31 a

Stage 10

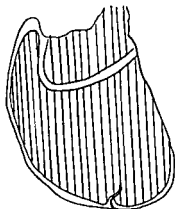


Fig 3 a



Fig 37 a

**Series B «small fragment»**

**Stage 1 (series B)**

This stage is the situation immediately after operation 1 for details see page 22 and Fig. 38





Fig. 38

### Stage 2 (series B)

This stage is represented by an animal sacrificed 53 days after operation

*Microscopic findings* The fragment lies in its concavity (Fig 39) The cleavage between fragment and concavity is distinctly visible Medially it is bridged by vascular connective tissue originating in the periosteum (Fig 41) The lateral portion of the cleavage contains primitive cartilage rich in cell In some places this shows an indistinct transition into the surrounding cartilage while in others there is a sharp line of demarcation (Fig 40) The ossification of the epiphysis has proceeded so far that the hnge and the proximate parts of the fragment have ossified The primitive cartilage in the lateral portion of the cleavage shows signs of ossification (Fig 40)

The roentgenogram shows a concavity in the outline of the subchondral bone (Fig 42)

The macroscopic findings are shown in Fig 43



Fig 39



Fig 40

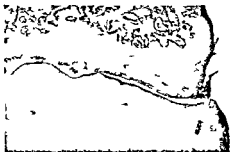


Fig 41



Fig 42

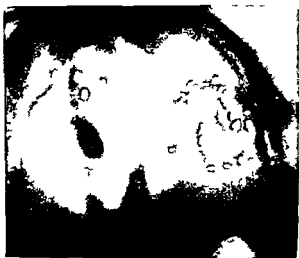


Fig 43

### Stage 3 a (series B)

This stage is represented by an animal sacrificed 82 days after operation

*Microscopic findings* The fragment lies in its concavity and the cleavage between fragment and concavity is distinctly visible. Medially it is bridged by vascular connective tissue originating in the periosteum (Fig. 44). The subchondral zone of ossification is separated from the cleavage by a thin layer of hyaline cartilage. Laterally there is a portion where the bone trabeculae are covered only by vascular connective tissue rich in cells. The vessels of this tissue anastomose with the vessels of the underlying spongy bone (Fig. 43; note arrow). In the zone of ossification a concavity corresponding to the experimental lesion is formed. In the medial portion of the fragment there is a bony nucleus (Fig. 45) with marrow cavities containing erythrocytes, living bone trabeculae and osteoid tissue. The trabeculae are well stained and unbroken; the lacunae contain cells; the walls of the blood vessels are intact. This centre of ossification is supplied via the vascular connective tissue which has developed *postoperatively* and bridges the medial portion of the cleavage (Fig. 45).

The *roentgenogram* shows a concavity in the outline of the bone and in this there is a separate bone shadow (Fig. 46).

The *macroscopic findings* are shown in Fig. 47.



Fig. 44



Fig. 45



Fig. 46

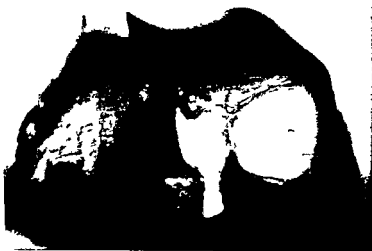


Fig. 47

### Stage 3 b (series B)

This stage is represented by an animal sacrificed 109 days after operation.

*Microscopic findings* The fragment lies in its concavity and the cleavage is distinctly visible. It is lined with hyaline cartilage. In the zone of ossification a concavity is formed under the fragment (Fig. 18). To its major portion the fragment has ossified and its bone tissue is directly connected with the rest of the epiphyseal bone via the ossified hinge. The bone structure of the fragment is dense with thick trabeculae and narrow marrow cavities. The latter contain erythrocytes. There are no cells in the lacunae (Fig. 19). The bone tissue seems necrotic.

The roentgenogram shows a concavity in the outline of the subchondral bone. In this a separate bone shadow is seen (Fig. 50).

The macroscopic findings are shown in Fig. 51.



Fig 48



Fig 49



Fig 50



Fig 51

### Stage 4 a (series B)

This stage is represented by an animal sacrificed 112 days after operation

The fragment is hanging from a pedicle of ligamentous tissue in the intercondylar fossa (Fig. 56)

*Microscopic findings* In the fragment a centre of ossification is situated eccentrically near the point of attachment of the ligamentous peduncle (Fig. 52) The cells in the lacunae of the ossific centre are well stained in some parts while in others they are indistinct The marrow cavities contain erythrocytes and osteoblasts The bone and osteoid trabeculae are whole Thus the bony nucleus of the fragment shows slight signs of decreased vitality although necrosis is not demonstrable (Fig. 54) In the subchondral zone of ossification of the medial condyle a shallow concavity is seen This is covered by hyaline connective tissue the deeper layers of which show a gradual transition to primitive cartilage The latter is in some parts separated from the subchondral zone of ossification by a thin layer of hyaline cartilage the cells of which are arranged in dispersed clusters (Fig. 53) Both these cartilaginous structures show signs of enchondral ossification

The roentgenogram shows a concavity in the outline of the bone of the medial condyle In this a separate bone shadow is seen (Fig. 55)

The macroscopic findings are shown in Fig. 56



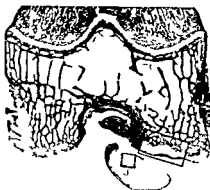


FIG 52



FIG 53

FIG 54

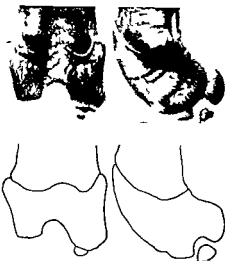


FIG 55



FIG 56

### Stage 4 b (series B)

This stage is represented by an animal sacrificed 132 days after operation

The fragment is found as a detached loose body in the joint cavity as is seen in Fig. 61 (note arrow) which is a roentgenogram of the knee joint of the animal *in vivo*

*Microscopic findings* The fragment consists of bone surrounded by hyaline cartilage. The lacunae and marrow cavities are empty and the bone trabeculae are broken, which suggests that the bone tissue is necrotic. The structure is dense (Figs 59 and 60). The medial condyle shows a concavity involving both the joint cartilage and the underlying bone. In the lateral portion of the concavity the bone is covered by a thick layer of hyaline cartilage (Fig. 57). More medially the bone trabeculae are in some places covered with a fragmentary layer of hyaline cartilage, in others by primitive cartilage towards the surface showing a gradual transition to hyaline connective tissue. At the medial border of the concavity the bone trabeculae are covered only by fibrous connective tissue (Fig. 58).

The *roentgenogram* shows a concavity in the outline of the bone of the medial condyle and a fragment with distinct bony structure separated from the concavity (Fig. 61).



Fig. 57



Fig. 58

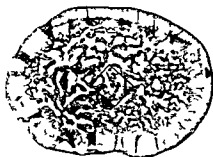


Fig. 59



Fig. 60

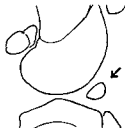
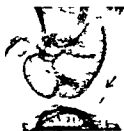


Fig. 61

### Summary of the results in series B

The experimental lesion showed a uniform course of development in the 12 animals first killed (up to 53 days postoperatively) of the total number of 40 in this series. This initial phase is described as stages 1 and 2.

In the remaining 28 animals sacrificed at various intervals from 57 to 200 days postoperatively, three different lines of development seemed to be discernible.

1. The fragment ossified but was detached so that a pedunculated or non-pedunculated loose body resulted which contained a bony nucleus. This was either living or necrotic. The fragment was supplied either via the hinge left at operation or via a connective tissue bridge which developed postoperatively over the medial portion of the cleavage. In some cases the bony nucleus of the fragment showed a strikingly dense structure. The concavity in the medial condyle was covered in part by hyaline cartilage in part by primitive

## PLATE IV

Stage 1



Fig 38 a

Stage 2

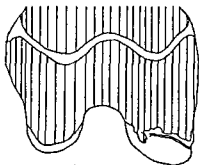


Fig 39 a



Fig 42 a

Stage 3 a

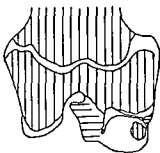


Fig 44 a



Fig 46 a

Stage 3 b

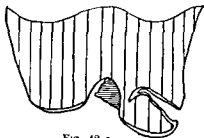


Fig 48 a



Fig 50 a

cartilage showing a gradual transition to hyaline cartilage and in part by fibrous connective tissue alone. These layers of cartilage underwent enchondral ossification. This line of development was observed in 5 animals and was described as stages 3 a, 3 b, 4 a and 4 b.

2. The fragment ossified and fused with the underlying bone. This line of development was not illustrated by examples since the course was the same as in series A, large fragments. This group seemed to comprise 8 animals.

3. The fragment did not ossify but was detached and formed a loose body in the joint cavity consisting entirely of cartilage. No examples from this group were described since the course was the same as in series D, loose fragment. This group seemed to comprise 15 animals.

See Plates IV and V. The figures in the plates (denoted a) are contour drawings of micrographs and roentgenogram with corresponding numbers shown in connection with the description of the various stages.

## PLATE V

Stage 4 a

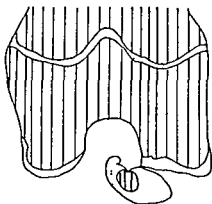


Fig 52 a



Fig 53 a

Stage 4 b

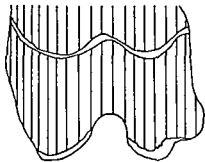


Fig 54 a



Fig 61 a

cartilage showing a gradual transition to hyaline cartilage and in part by fibrous connective tissue alone. These layers of cartilage underwent enchondral ossification. This line of development was observed in 5 animals and was described as stages 3 a, 3 b, 4 a and 4 b.

2 The fragment ossified and fused with the underlying bone. This line of development was not illustrated by examples since the course was the same as in series A (large fragments). This group seemed to comprise 8 animals.

3 The fragment did not ossify but was detached and formed a loose body in the joint cavity consisting entirely of cartilage. No examples from this group were described since the course was the same as in series D (loose fragment). This group seemed to comprise 15 animals.

See Plates IV and V. The figures in the plates (denoted a) are contour drawings of micrographs and roentgenograms with corresponding numbers shown in connection with the description of the various stages.





H. C



H. C

### Stage 2 (series C)

This stage is represented by an animal sacrificed 22 days after operation

*Microscopic findings* The subchondral zone of ossification is similarly arched in the medial and lateral condyles. The fragment hangs upon its hinge of ligamentous tissue (Fig. 64). The cartilage cells stain identically throughout the fragment (Fig. 65). At the former site of the fragment in the medial condyle a concavity is formed, at the bottom of which a layer of primitive cartilage with closely packed cells is seen. This is separated from the underlying normal cartilage by a narrow zone of cartilaginous matrix devoid of cells (Fig. 67).

The *macroscopic findings* are shown in Fig. 66.



Fig 64



Fig 65



Fig 66

### Stage 3 a (series C)

This stage is represented by an animal sacrificed 63 days after operation

*Microscopic findings* The ossification of the condyle has proceeded so far that a concavity has formed in the subchondral bone at the former site of the fragment (Fig. 67). In the joint surface a corresponding concavity is seen. The bottom of the latter consists of cartilage, which here and there in the proximity of the subchondral zone of ossification shows dispersed clusters of cartilage cells. Other parts of the concavity are lined with primitive cartilage rich in cells. Both types of cartilage show signs of enchondral ossification (Fig. 68).

The fragment hangs from a hinge of ligament tissue. In the centre of the cartilaginous fragment there are distended cells with relatively small nuclei and an abundance of faintly stained matrix is seen. The superficial parts of the fragment have a normal appearance (Fig. 68).

The *roentgenogram* shows a shallow concavity in the outline of the bone of the medial condyle (Fig. 69). The fragment gives a weak shadow indicating calcium content but lack of bony structure.



Fig. 6



Fig. 6A



Fig. 6J

### Stage 3 b (series C)

This stage is represented by an animal sacrificed 172 days after operation. The fragment hangs from a hinge of ligament tissue attached to the intercondylar fossa (Fig. 7) the arrow indicates the concavity).

*Microscopic findings* In the operated condyle the subchondral zone of bone shows a shallow concavity at the former site of the fragment (Fig. 70). Over this the joint cartilage is thin and hyaline and shows an irregular structure with densely arranged cells. At this site the joint surface is uneven. In the proximity of the intercondylar fossa the bone trabeculae are covered by a thin layer of fibrous connective tissue (Fig. 72). In the cartilage of the fragment near the point of attachment of the ligamentous peduncle a bony nucleus is seen (Fig. 71). In the lacunae of the latter there are well stained bone cells and the marrow cavities contain erythrocytes. The cartilage of the fragment shows degenerative changes and is covered very superficially by a layer of fibrous connective tissue (Fig. 73).

The roentgenogram shows that the two condyles have similar curved outlines. The fragment gives rise to a shadow which laterally is indicative of bony structure (Fig. 74).

The macroscopic findings are shown in Fig. 75.



Fig. 0



Fig. 1



Fig. 2



Fig. 3



Fig. 4



Fig. 5

### Summary of the results in series C

In 17 of the 18 animals in this series the lesion seemed to develop as was described in the foregoing (stages 1—3 a)

During the course of development the fragment was dislocated from its crater. Under the latter a corresponding concavity was formed in the zone of ossification. The crater was incompletely filled with primitive cartilage which later was replaced by hyaline cartilage of somewhat irregular structure. Most of this cartilage ossified so that the concavity in the subchondral zone of bone was in part levelled out. The operated condyle remained deformed as compared with the intact condyle.

The fragment grew hanging from its ligamentous peduncle. On the surface its cartilage maintained a normal structure in the middle it showed degenerative change and at the end of the observation time it was calcified. The fragment was surrounded by a thin capsule of connective tissue. It remained attached to its peduncle of ligament tissue.

In one case a living bony nucleus was found in the pedunculated fragment. It was supplied by vessels in the ligamentous pedicle (stage 3 b).

See Plate VI. The figures in the plate (denoted a) are contour drawings of micrographs and roentgenograms with corresponding numbers shown in connection with the description of the various stage.



## PLATE VI

Stage 1



Fig 63 a

Stage 2

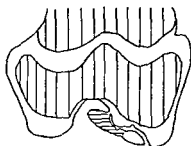


Fig 64 a

Stage 3 a

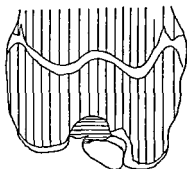


Fig 65 a



Fig 69 a

Stage 3 b

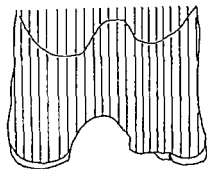


Fig 70 a



Fig 74 a

## Series D «loose fragment»

### Stage 1 (series D)

This stage is represented by an animal sacrificed 8 days after operation (For details regarding the experimental lesion see page 22 )

*Microscopic findings* In the cartilage of the condyle a defect is seen (Fig 76) The joint cartilage in the proximity of the concavity shows ill stained cartilage cells

The fragment which lies as a loose body in the joint cavity shows well stained cartilage cells of normal size and shape in all parts of the cross section Its periphery consists of a single layer of flat connective tissue cells (Fig 77)



Fig. 6



Fig. 7

### Stage 2 (series D)

This stage is represented by an animal sacrificed 26 days after operation.

*Microscopic findings* There is a concavity in both the joint surface and the underlying zone of ossification (Figs 75). In the concavity of the joint surface a layer of primitive cartilage rich in cells is seen. This shows no signs of enchondral ossification. The transition to normal joint cartilage is in some parts indistinct, in others distinct and characterized by a streak of ill stained cartilage cells (Fig 80).

Most of the fragment which lies as a loose body in the joint cavity consists of normal looking cartilage. In the centre of the fragment there are occasional cell lacking nuclei. On its extreme periphery the fragment is surrounded by a thin capsule of fibrous connective tissue and under this there is a layer of cartilage of irregular structure rich in cells (Figs 79 and 81).

The *roentgenogram* shows a concavity in the outline of the bow of the operated condyle (Fig 82). The fragment gives no shadow.



Fig. 78

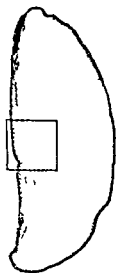


Fig. 79

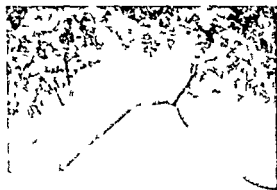


Fig. 80



Fig. 81



Fig. 82

### Stage 2 (series D)

This stage is represented by an animal sacrificed 26 days after operation.

*Microscopic findings* There is a concavity in both the joint surface and the underlying zone of ossification (Fig 78). In the concavity of the joint surface a layer of primitive cartilage rich in cells is seen. This shows no signs of enchondral ossification. The transition to normal joint cartilage is in some parts indistinct in others distinct and characterized by a streak of ill stained cartilage cells (Fig 80).

Most of the fragment which lies as a loose body in the joint cavity consists of normal looking cartilage. In the centre of the fragment there are occasional cells lacking nuclei. On its extreme periphery the fragment is surrounded by a thin capsule of fibrous connective tissue and under this there is a layer of cartilage of irregular structure rich in cells (Figs 79 and 81).

The *roentgenogram* shows a concavity in the outline of the bone of the operated condyle (Fig 82). The fragment gives no shadow.

Fig. 83



Fig. 82



Fig. 84

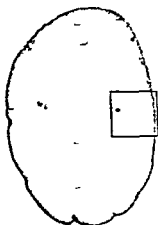


Fig. 86



Fig. 87



### Summary of the results in series D

In all the 16 animals of the series the course of development seemed to be that which was described in the foregoing as stages 1—4

The concavity in the cartilage was incompletely filled out with primitive cartilage which was later replaced by hyaline cartilage of irregular structure. This cartilage ossified slowly so that a concavity was initially formed in the subchondral zone of ossification. As ossification proceeded this concavity was levelled out. At the end of the observation time the condyle was somewhat deformed inasmuch as it showed a shallow concavity at the site where the fragment had been gouged out. The cartilage at the centre of the fragment degenerated and calcified while at the end of the observation time the peripheral parts still showed signs of vitality and at certain sites an irregular structure and densely packed cells. Most superficially the fragment was surrounded by a thin connective tissue capsule, which may suggest that the increase in volume of the fragment in part at least was due to appositional growth.

See Plate VII. The figures in the plate (denoted a) are contour drawings of micrographs and roentgenograms with corresponding numbers shown in connection with the description of the various stages.



Stage 1



Fig. 5 a

Stage 2

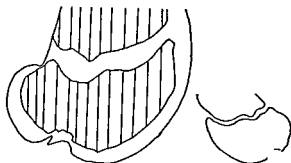


Fig. 58 a

Fig. 52 a

Stage 3

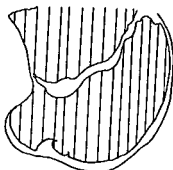


Fig. 53 a

Stage 4

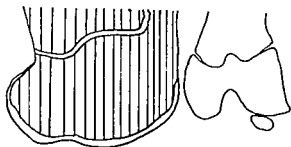


Fig. 54 a

Fig. 55 a

**Series E «intracartilaginous cleavage»****Stage 1 (series E)**

This stage is the situation immediately after operation. For details see page 22 and Figs 93 and 94

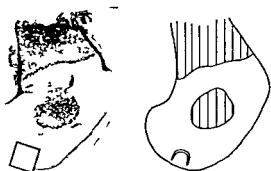


Fig 93

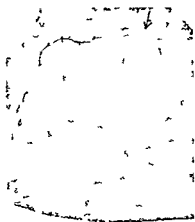


Fig 94

### Stage 2 (series E)

This stage is represented by an animal sacrificed 32 days after operation.

*Microscopic findings* The ossification of the epiphysis has proceeded so far that a concavity has formed in the subchondral zone of ossification at the site of the intracartilaginous cleavage (Fig. 95). The cleavage is almost completely filled by primitive cartilage which in some parts is separated from its surroundings by a more intensely stained streak in the matrix without any cells in the proximity and which in other parts shows a gradual transition (Fig. 96). In certain places the cleavage is visible. At the e the cartilage of the separated piece and the thin layer of cartilage covering the zone of ossification meet (Fig. 96). The cartilage cells at the centre of the separated piece are distended and their nuclei are pyknotic (Fig. 96).

The roentgenogram shows a concavity in the outline of the bone of the medial condyle (Fig. 97).



Fig. 92



Fig. 96



Fig. 9

### Stage 3 (series E)

This stage is represented by an animal sacrificed 35 days after operation.

*Microscopic findings* In the zone of ossification of the medial condyle a concavity is seen. In the piece separated by the intracartilaginous cleavage there is a living bony nucleus (Fig. 98). The cleavage contains some granulation tissue the vessels of which anastomose with the vessels of the separate bony nucleus and with the vessels of the spongy bone of the epiphysis. The bony nucleus of the separated piece is surrounded by a layer of hyaline cartilage which in some places narrows to a thin border of cartilaginous matrix containing scattered clusters of cartilage cells. The cleavage is almost completely filled by an irregular layer of hyaline cartilage with scattered cells. In some places the cleavage is visible and contains a picrinophilic structureless substance. The zone of ossification of the concavity is lined with a layer of more intensely stained cartilaginous matrix with scattered clusters of cartilage cells (Fig. 99).

The roentgenogram shows a concavity in the outline of the subchondral bone. In the concavity there is a separate bone shadow which in part is clearly distinguishable from the surroundings (Fig. 100).



Fig. 95



Fig. 96



Fig. 100

### Stage 4 (series E)

This stage is represented by an animal sacrificed 64 days after operation.

*Microscopic findings* In the spongy bone of the medial condyle a transverse curved streak of indeterminate structure is seen. In this and in its surroundings osseoid tissue is observable (Fig. 101 and 102). As a continuation of the streak under the medial periosteum of the condyle there is thickened hyaline cartilage (Fig. 101). Around the streak of osseoid tissue the bony structure is denser as compared with that of the lateral condyle (Fig. 101).

The *roentgenogram* shows minor irregularities in the bony structure of the operated condyle (Fig. 103).





Fig. 101

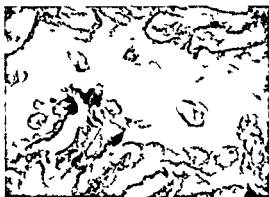


Fig. 102



Fig. 103

### Summary of the results in series E

In all the 18 animals of this series the course of development of the experimental lesion seemed to be as described in the foregoing stages I—II.

In the intracartilaginous cleavage primitive cartilage was formed which later was succeeded by hyaline cartilage. At the site of the experimental lesion ossification was arrested so that a concavity was formed in the zone of ossification. At the centre of the cartilaginous piece which was separated by the intracartilaginous cleavage degenerative changes and later a separate bony nucleus developed. The latter was supplied in part by vessels which originated in the perichondrium (the periosteum) and bridged the medial opening of the cleavage in part by vessel which reached the separated piece via granulation tissue present at the bottom of the cleavage.

By enchondral ossification the hyaline cartilage which developed in the cleavage was divided into fragments. Thus islands of hyaline cartilage were formed in the spongy bone. These islands too underwent enchondral ossification and in the course of development a normal bony structure was reestablished although at the end of the observation time the operated condyle was somewhat broader than the non-operated condyle.

See Plate VIII. The figures in the plate (denoted a) are contour drawings of micrographs and roentgenograms with corresponding numbers shown in connection with the description of the various stages.

## PLATE VIII

Stage 1

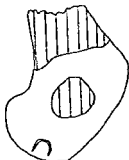


Fig 93 a

Stage 2

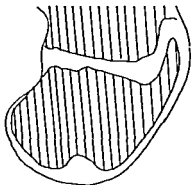


Fig 95 a



Fig 97 a

Stage 3

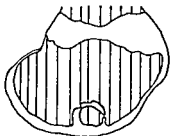


Fig 98 a



Fig 100 a

Stage 4

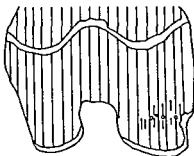


Fig 101 a



Fig 103 a

## DISCUSSION AND CONCLUSIONS

### The present results in the light of the relevant literature

In series A large fragments the cleavage between fragment and concavity initially contained fibrin then granulation tissue then hyaline connective tissue succeeded by primitive cartilage, and finally hyaline cartilage. This is in agreement with the observation made by SHANNON (1931) who performed experiments on dogs (probably adult dogs, the age was not indicated). The origin of the regeneration observed is not revealed by the present study. RAO (1954) and GIBSON (1955) stated that hyaline cartilage regenerates in three different ways: 1) by proliferation of chondrocytes, 2) by proliferation of vascular connective tissue originating in the perichondrium and 3) by invasion of vascular connective tissue from the subchondral bone. It appears that in an attempt to account for the present results the first two alternatives may be considered but not the third alternative since the subchondral bone was left intact at operation. On the other hand it is probable that the vessels in the joint cartilage were injured and it is possible that the regeneration originated in the endothelium of these.

It appears that in series A the ossification of the fragment originated in vessels penetrating from the granulation tissue present in the cleavage between fragment and concavity. It is also possible however that the ossification of the fragment was due to the penetration of vessels from the *intercondylar fossa*. These vessels may have existed before operation and remained intact (Cf series A stage 3 Fig. 9).

Figs 28 and 33 in series A stages 9 and 10 showed that the superficial layer of the joint cartilage regenerated in such a way that the cleavage was filled with hyaline cartilage which was distinguishable from the surrounding normal cartilage only by a somewhat irregular arrangement of the cartilage cells. This tallies with the observations made by GIES (1883), NAGURA (1937), NAGURA & KOSUGA (1938) and CARLSON (1957).

After incision and subchondral infraction respectively of epiphyseal bone in growing rabbits NAGURA (1938) and BURCKHARDT (1948) observed

that the subchondral defect was filled with a cartilage-like tissue. This tissue showed very low enchondral ossification and may perhaps be compared with the primitive cartilage which in the present study was found in the cleavage between fragment and cartilage and ossified much later than the cartilage of the fragment (cf. series A, B and E).

In growing rat BRAHEAR (1949) created a transverse fracture through the cartilage of the epiphyseal plate at the distal end of the femur. At some distance from the fracture the ossification of the portion of the epiphyseal plate situated proximally to the fracture was arrested for a long time. In the portion of the epiphyseal plate which was distal to the fracture a secondary bony nucleus developed after vascularization. Only later did the portion of cartilage bordering on the fracture line undergo ossification. Although this experiment involved the epiphyseal plate it has certain features in common with the results in series A and I in the present study. In both cases a cleavage was made in cartilage which normally ossifies and in both cases a separate bony nucleus developed distally to this cleavage.

In series A and B it appeared that the fragments ossified first at the site where the invading vessel penetrated into the fragment and not at the site where the cartilage cells could be assumed to be oldest, i.e. proximally to the bottom of the concavity. Proximally to the cleavage between fragment and concavity the cartilage ossified relatively late. This was correspondingly observed in series L (intracartilaginous cleavage). The phenomenon shows features in common with those noted by BRASHAR (1950). Why the cartilage bordering on the cleavage ossified relatively late remains an open question.

ENHENVORGB, ENFELDT and OLSSON (1961) tore the ligamentous attachment to the cartilaginous tibial tuberosity in 1 month old dogs. In one case a radiopaque tissue fragment was observed separately from the apophyseal bony nucleus of the tibial tuberosity which exhibited a defect corresponding to the fragment. It may be supposed that this phenomenon was analogous to those demonstrated in this study in series A, B and E.

According to STRANGEWAYS (1920), FISHER (1922) and LANDELLS (1957) articular cartilage is supplied by the synovial fluid. LANDELLS stated that cartilage may live, grow and calcify (but not ossify) when completely detached from bone provided it has free access to the synovial fluid. This theory is substantiated by the present results in series D (loose fragments) inasmuch as none of the cartilaginous fragments which were initially completely detached from their concavities ossified. They grew

showed signs of vitality and calcified. In series B small fragments it was found that the fragment was sometimes vascularized and ossified via the hinge of cartilage and ligament tissue which was left at operation or via a connective tissue bridge which developed *postoperatively* and originated in the periosteum at the medial margin of the condyle and bridged the medial portion of the cleavage. This finding contradicts LANDELL'S assertion to some extent since it implies that growing articular cartilage may live, grow and calcify and in the presence of a vascular connection also ossify although the cartilage is completely separated from bone (Cf. DAVIS & LABARRE 1957 and LACROIX 1959 see below).

DAVIS & LABARRE (1957) and LACROIX (1959) excised a piece of cartilage from the head of the radius of a new born monkey and from fracture callus respectively and implanted the fragment into the eye or under the renal capsule in adult animals. The fragments ossified. The implantation of costal cartilage in a similar way brought about only slight and retarded ossification. Under normal conditions joint cartilage from a new born animal and cartilage from fracture callus ossifies while costal cartilage does not ossify. The cartilaginous fragments too which in the present study were partially detached from the joint surface and which ossified would also normally have ossified. This shows that cartilage which normally ossifies during the course of development may also maintain a tendency towards ossification when detached from direct continuity with its original surroundings (See series A, B and C).

In series B it was found that some fragments contained a bony nucleus showing a denser structure than normal (cf. Figs. 48 and 59). BODECHKO & HARRIS (1960) detached the head of the femur in adult rabbits, severed the round ligament and nailed the head of the femur into place again. Initially they observed necrosis of the bone trabeculae in the head of the femur and subsequently apposition of new bone to the necrotic trabeculae corresponding to a diminution of the marrow cavities and a denser shadow in the roentgenogram. Similar observations were made by GAMMERN & VENTURINI (1959) and by RUTH HAUSER, ROHNER & HELD (1960). It is possible therefore that the dense structure of the spongy bone observed in certain fragments in the present study was due to poor nutrition, necrosis of the bony portion of the fragment and reossification as in the experiment described by BODECHKO & HARRIS.

In the available literature the author has not encountered any report except LANCKENSHILDS (1955) to the effect that a partially detached piece of joint cartilage in a growing individual may result in a pedunculated joint.

mouse with a nucleus of necrotic or living bone as was found in the present study series B. In this a similar lesion gave rise also to a completely loose joint mouse with a nucleus of necrotic bone.

In series B certain fragments fused with the surrounding bone tissue at the site from which they had been gouged out just as in series A. Others ossified and were then detached, still others were detached without foregoing ossification. In series A all fragments underwent bony union with their bed. This difference may be due to the fact that in series A the fragments were relatively deeper (1.4—1.8 mm) in relation to their extension in the plane of the joint surface than in series B (0.7—1.2 mm). Hence it would seem that the mechanical conditions for the fragment remaining in the concavity were more favourable in series A than in series B. On the other hand in series A the lesion extended more deeply towards the bony nucleus of the epiphysis than in series B. Hence the risk was probably greater that the vessels in the cartilage (which probably are more abundant near the bony nucleus than near the surface) would be injured in series A. It is possible that granulation tissue originating in injured vessels in the cartilage contributed to the fixation of the fragment to its bed.

In series D also the fragments it was found that during the course of development the fragment degenerated at the centre and showed signs of proliferation peripherally. The fact that toward the end of the observation time the fragment gave a relatively dense shadow in the roentgenogram suggests that calcium had been deposited. Around the fragment a thin capsule of connective tissue was formed. All the findings are in agreement with the observations made by GIES (1882), STRANGEWAYS (1920), PHEMISTER (1931), BENNET BAUER & MADDOCK (1932) and LANDALLS (1937).

In series D the cartilage bordering on the operatively created defect showed cartilage cell with markedly diminished stainability soon after operation. A similar observation was made by CARLSON (1937) who detached a small chip of cartilage from the joint surface of the patella in growing rabbit.

The defect in the joint cartilage created in series D was filled by primitive cartilage which later was succeeded by hyaline cartilage with a partially striated matrix. BENNET BAUER & MADDOCK (1932) reported that after an analogous operation on adult dog the end result was fibrous cartilage.

BENNET BAUER & MADDOCK (1932) and BUCHER (1933) gouged out a fragment of cartilage together with a piece of underlying bone. The tissue filling the concavity (fibrous cartilage according to BUCHER) ossified in its deeper portions. In the present series D a concavity developed in the

zone of ossification at the site of the experimental lesion (see Fig 78 page 77) before the cartilage which filled the concavity in the cartilage ossified in its deeper layers (see Fig 83 page 79 and Fig 88 page 81)

In series C change of ligament tissue the results were in agreement with the results in series D loose fragments with regard to the development of both the fragment and the concavity. In one case however in series C a bony nucleus developed in the fragment (Fig 71 page 71). Obviously this bony nucleus was supplied by vessels in the hyaline peduncle but when it developed is less obvious. The concavity in the joint cartilage at the former site of the fragment was partially levelled out and covered by hyaline cartilage which only differed slightly from intact articular cartilage. Hence it may be concluded that the fragment had been dislocated from the concavity relatively early. In addition the bony nucleus was relatively small in relation to the whole fragment and its osteoid trabeculae were relatively narrow and contained living cells (Fig 72 page 71). This seems to suggest that the centre of ossification had developed relatively late. The implication would be that *the centre of ossification developed after the fragment had been dislocated from its bed*. This hypothesis is not however substantiated by any observations reported in the literature nor does it account for the fact that no bony nucleus developed in any other fragments in this series.

In the available literature no data were found concerning experiments involving an intracartilaginous cleavage in growing joint cartilage. In series E intracartilaginous cleavage the course was practically the same as in series A although in series E the superficial layer of the joint cartilage had been left intact. The results in series F showed that an intracartilaginous cleavage too in growing joint cartilage may later give rise to a separate centre of ossification.

RIBBING (1951), CAFFEY, MADILL, ROYER & MORALES (1958) and others have emphasized the occurrence of accessory bony nuclei *e.g.* in the distal femoral epiphysis in children. The same phenomenon has been observed in other epiphyses by ROCHT & SUNDERLAND (1959). These accessory bony nuclei were so common that the last mentioned writers claimed that they *should be regarded as normal*. CAFFEY *et al.* (1958) detected accessory bony nuclei in the distal femoral epiphysis in children aged between 1 and 12 years in 19 cases out of 147. RIBBING (1951) and SMILLIE (1960) suggested that some of these accessory centres of ossification are precursors to juvenile osteochondritis dissecans. The results in the present series E showed that an accessory bony nucleus in a growing individual may result from an intra-



cartilaginous cleavage sustained at an earlier age. Whether a condition resulting from a previously sustained cleavage in cartilage was involved in some of the cases of accessory bony nuclei in children described by the above mentioned writers remains an open question.

### The present results as compared with the pathological anatomy of osteochondritis dissecans

ROBERTS (1957) stated with regard to osteochondritis dissecans "No hard and fast criteria can be laid down as being necessary to justify the diagnosis but it is usually accepted that the diagnosis can be properly applied to any case in which a portion of articular surface not necessarily containing bone separates or attempts to separate by a process of gradual and dissecting extrusion." Since in the present study both completely loose and pedunculated joint mice some of them containing a bony nucleus and others not were experimentally created there seems to be a resemblance between the present results and osteochondritis dissecans if ROBERTS' definition of this condition is accepted.

A common finding in osteochondritis dissecans is that the border of the bony nucleus is surrounded by hyaline cartilage extending for some distance between the fragment and the concavity (LEB 1924 LEHMANN 1925 LACROIX 1941 BIANCHI *et al* 1955). A similar observation was made in several experiments in the present study (Fig 22 page 37 Fig 25 page 39 Fig 34 page 43 and Fig 34 page 47).

LACROIX (1941) described a case of osteochondritis dissecans in which the outer surface of the fragment was coated with normal cartilage which at the margin included the periphery of the underlying bony nucleus (Fig 104). On the aspect facing the concavity the bony nucleus was covered by what LACROIX called young cartilage which was characterized by the arrangement of cells in clusters (Fig 104 b). In stage 7 series A of the present study (Fig 22 page 37) and in stage 4 series B (Fig 34 page 47) a similar finding was made at the corresponding site of the fragment.

In the case described by LACROIX the cartilage cells at the margin of the fragment were arranged in rows at right angles to the articular surface and parallel with the cleavage (Fig 104 a). A similar finding was made in series A stage 10 (Fig 34 page 43).

PHENISTER (1924) LEHMANN (1925) and BIANCHI *et al* (1955) described cases of osteochondritis dissecans in which the spongy bone of the frag-



) Fig. 104



) Fig. 104 b



) Fig. 104 a

ment showed a strikingly dense structure (Fig. 105). There was a similar finding in series B of the present study stage 3b (Fig. 49 page 55) and stage 4b (Fig. 59 and 60 page 59).



) Fig. 105

In series A and B many of the separate fragments united with their bed. Analogously in series E this was consistently the case. Spontaneous

) (With the courtesy of F. LACROIX)

) (With the courtesy of J. C. LEHMANN)

healing of the lesion in osteochondritis dissecans has been reported by WIBERG (1943) NOVOTNY (1951, 1952) PICK (1955) and others.

On roentgenological follow up of patients with osteochondritis dissecans FREIBERG (1923) and SCHELLER (1960) observed that the shadow of the



B

Fig. 106 A Anteroposterior view of the knee of a 16-year-old boy with osteochondritis dissecans. B The same knee two years later. Note the growth of the fragment (A LANGENSSKJÖLD unpublished observation).

fragment grew. In the Orthopaedic Hospital of the Invalid Foundation growth of the fragment has been observed in several cases of osteochondritis dissecans (A LANGENSKIÖLD unpublished observations). See Fig 106. That growth of the fragment occurred in the present study series A could be inferred from some of the micrographs (Fig 11 page 31 Fig 14 page 33 and Fig 17 page 35). SCHELLER explained his observation as not an expression of authentic growth but of increased calcium content in the lesion which is therefore visualized more distinctly on the roentgenogram. It is also possible however that the roentgenological finding reported by FREIBERG SCHELLER and LANGENSKIÖLD may be the manifestation of a process analogous to that described in the present series A i.e. to authentic growth.

PHENISTER (1924) and FAIRBANK (1933) pointed out that in osteochondritis dissecans fragments which originated in the lateral margin of the condyle and remained pedunculated by ligament and synovial tissue contained living bone and showed signs of bone formation. A similar finding was made in the present study series B, stage 1a (Fig 54 page 57) and series C stage 3b (Fig 71 page 71).

LEHMANN (1925) described a case of osteochondritis dissecans in which downward protrusion of the joint cartilage marked the border between the fragment and its surroundings (Fig 107 note arrow). A similar finding



Fig 107

was made in the present series A stage 9 (Fig 28 page 41). In addition LEHMANN observed islands of hyaline cartilage at the border between the fragment and its bed. A similar feature was observed in the present study series A stages 8 and 9 (Fig 25 page 39 and Fig 27 page 27).

Several authors (see page 19) have shown that the bony nucleus of the fragment was visible in osteochondritis dissecans. Living bone in the separate fragment was also observed in many cases in the present study.

1 (With the courtesy of J. C. LEHMANN)

LEHMANN (1923) FAIRBANK (1933) and others have reported that the concavity remaining in the articular surface after separation of the fragment was lined with fibrous connective tissue fibrous cartilage or hyaline cartilage. *In the present study a similar finding was repeatedly made at the corresponding site (Fig 45 page 53 Fig 48 page 55 Fig 53 page 57 Fig 58 page 59 and Fig 73 page 71)*

### **The present results as compared with the data on experimentally induced imitations of osteochondritis dissecans**

As mentioned in the survey of the literature many investigators have tried to create osteochondral joint mice in laboratory animals by detaching pieces of cartilage and bone completely or partially from the joint surface and replacing them in their beds. The fragments united with the joint capsule with the site from which they had been gouged out with the synovial tissue or the periosteum or were completely resorbed.

In order to study the uptake of radioactive sulphur in loose joint mice BAILEY & SELLE (1959) excised a fragment (probably consisting of cartilage and bone) from the surface of the knee joint in 4 to 8 week old rabbits and placed it in the intercondylar fossa. After 2 to 6 months the fragments were found as loose bodies with necrotic bony nuclei surrounded by fibrous connective tissue. The concavity at the site from which the fragment had been excised was covered by fibrous connective tissue. The writers performed no experiments in which the fragments were replaced in their beds.

By repeated blunt trauma of the joint surface REHBEIN (1950) brought about necrosis of the cartilage and the underlying bone. The necrotic area was separated from the surroundings by a streak of loose tissue. At the proximal side of this bone formation was seen at the distal side reorption of bone. Loose bodies did not result.

By incision or infraction of articular cartilage and the underlying bone in growing rabbits NAGURA (1938) and BURCHARDT (1948) brought about the development of a streak of hyaline substance which underwent slow enchondral ossification. In this way NAGURA succeeded in separating a piece of the subchondral bone of the patella. Loose bodies did not result.

In this connection the following statements by DE PALMA (1954) concerning the etiology of osteochondritis dissecans may be cited. "Critical assessment of the varied theories advanced by numerous investigators fails to provide an explanation for the origin of loose bodies under discussion

which is supported by indisputable clinical and scientific observation. Although most investigators feel that trauma in some form or manner plays a significant role, no foolproof mechanism has yet been conceived.

In the present study both completely loose and pedunculated joint mice both with and without a living or necrotic bony nucleus were created. Furthermore, a marked resemblance could be demonstrated between the results and the pathological anatomy of osteochondritis dissecans. It may perhaps be stated therefore that the present results imitate osteochondritis dissecans more obviously than any previously reported in the literature. Thus the hypothesis formulated in A. LANGE-SKJOLD's paper of 1933, "that osteochondritis dissecans arises as a sequel of a cartilage fracture in early childhood," seems to be justified.

## SUMMARY

In order to study more closely the development of the post traumatic condition resembling osteochondritis dissecans described by V. LANGENSKIÖLD in 1933 mechanical lesions of articular cartilage were created in a total of 114 rabbits aged from one to 12 days. On the basis of the type of experimental lesion induced the material was divided into the following five series:

*Series 1 (large fragments)* From the cartilage of the medial femoral condyle a fragment 1.4—1.8 mm deep was gouged out with a sharp scoop. A hinge of cartilage and ligament tissue facing the intercondylar fossa was excepted (Figs. 1 and 2, page 23).

The series comprised 20 animals killed at different intervals up to an age of 102 days.

*Series B (small fragments)* The same operation was made as in series A but the fragment was made only 0.7—1.2 mm deep (Fig. 38, page 49).

The series comprised 40 animals killed at different intervals up to an age of 200 days.

*Series C (hinge of ligament tissue)* The operation was performed as in series A but the hinge left consisted of ligament tissue alone (see Fig. 62, page 63).

The series comprised 18 animals killed at different intervals up to an age of 200 days.

*Series D (loose fragments)* The same operation was performed as in series A but the fragment was completely detached and replaced in its bed.

The series comprised 16 animals killed at different intervals up to an age of 120 days.

*Series E (intracartilaginous cleavage)* Using a special instrument an intracartilaginous cleavage was created in the cartilage of the medial femoral condyle. The cleavage separated a piece of cartilage from the underlying

tissue excepting the most superficial layer of the joint cartilage, which was left intact (see Fig 93 and 94 page 83)

The series comprised 18 animals killed at different intervals up to an age of 150 days

The specimens obtained were studied macroscopically microscopically and roentgenologically In the different series the following findings were made

*Series A large fragments* During the course of growth a concavity was formed in the subchondral zone of ossification (Fig 7 page 29) In the fragment a separate centre of ossification was formed which grew (Fig 11 page 31 Fig 14 page 33 and Fig 17 page 35) In the cleavage between the fragment and the concavity the following structures succeeded each other granulation tissue hyalinized connective tissue primitive cartilage and hyaline cartilage The cleavage disappeared and was replaced by a solid wall of hyaline cartilage (Fig 20 page 37) which was divided into fragments by enchondral ossification (Fig 27 page 41) and subsequently disappeared so that the previously separate bony nucleus fused with the rest of the epiphysis In the plane of the joint surface the cleavage persisted partially while partially its margins were united by hyaline cartilage showing an irregular structure (Fig 28 page 41 Figs 33 and 34 page 43)

*Series B small fragments* Three different lines of development were discernible

1 The fragment ossified and fused with its bed the course being mainly the same as in series A

2 The fragment ossified (Fig 41 page 53 and Fig 48 page 55) and became detached so that a completely loose (Fig 59 page 59) or pedunculated joint mouse (Fig 62 page 57) resulted which contained a necrotic or living bony nucleus The concavity was covered by fibrous connective tissue primitive cartilage or hyaline cartilage The two last mentioned tissues showed signs of ossification (Fig 63 page 57 and Fig 68 page 59)

3 The fragment did not ossify but became detached and formed a completely loose joint mouse The course was the same as in series B

*Series C change of ligament tissue* The fragment was dislocated from its bed Corresponding to the latter a concavity was formed in the zone of ossification (Fig 67 page 69) This concavity was partially filled by primitive cartilage which later was replaced by hyaline cartilage of somewhat irregular structure Most of this cartilage ossified so that the concavity in the outline of the bone was levelled out The condyle remained deformed (Fig 70 page 71) The fragment grew while remaining attached to the ligamentous peduncle On the surface its cartilage maintained its



normal structure. It was surrounded by a thin capsule of connective tissue. The central portions of the fragment degenerated and calcified. In one case the fragment contained a living bony nucleus (Fig 71 page 71).

*Series D «loose fragments»* The concavity in the cartilage was incompletely filled by primitive cartilage (Fig 79 page 77) later by hyaline cartilage of irregular structure (Fig 84 page 79). This cartilage ossified slowly so that a concavity initially was formed in the subchondral zone of ossification (Fig 79 page 77). Later this concavity was levelled out (Fig 88 page 81). The condyle remained deformed (Fig 92 page 81). The fragment grew, degenerated at the centre and calcified. Its peripheral parts remained viable.

*Series E «intracartilaginous cleavage»* In the intracartilaginous cleavage primitive cartilage and later hyaline cartilage was formed. A concavity developed in the subchondral zone of ossification (Fig 96 page 87). In the separated cartilaginous fragment a separate bony nucleus developed (Fig 99 page 89). Later the cartilage which had been formed in the cleavage ossified (Fig 101 page 91) and in the course of development normal conditions were re-established apart from the fact that the operated condyle remained broader than the intact one.

The results were assessed in the light of the relevant literature and compared with the data on the pathological anatomy of osteochondritis dissecans. Striking resemblances were found. The results of the present study seem to support the hypothesis formulated by A. LANGENSKIÖLD in 1933: «Can osteochondritis dissecans arise as a sequel of a cartilage fracture in early childhood?»

## REFERENCES

- ANHAUSEN G. Über den histologischen Vorgang bei der Transplantation Gelenkenden insbesondere über die Transplantation Gelenkknorpel und Epiphysenknorpel — *Langenbeck Arch Klin Chir* 99 1 111.
- Gelenkausbrüche und Gelenkknorpelbrüche im Tierversuch — *Langenbeck Arch Klin Chir* 124 343 1123.
- BAILEY B W & SEILL R L. A study of the growth and surgically created osteochondral loose bodies in adult and kits — *Surg Forum* 9 744 1958.
- BALANTYNE D I Jr & CONVERSE J M. Vascularization of connective tissue grafts transplanted to the chorion allantois of the chick — *Transplant Bull* 3 373 1958.
- BAUTH A. Die Entstehung und das Wachsthum der freien Gelenkkörper — *Langenbeck Arch Klin Chir* 36 507 1898.
- BAXTER H, GOLDSTEIN M A & McMILLAN G C. The fate of human homo- and heterografts — *Plast Reconstr Surg* 22 516 1958.
- BENNETT C A & BAUER W. Further studies concerning the repair of articular cartilage in dog joints — *J Bone Joint Surg* 17 141 1935.
- BENNETT C A, BAUER W & MADDOCK S J. A study of the repair of articular cartilage and the reaction of normal joints of adult dogs to surgically created defects of the articular cartilage — joint mice and patellar displacement — *Amer J Path* 38 499 1932.
- BENNINGHOFF A. Lehrbuch der Anatomie des Menschen — Urban & Schwarzenberg Berlin München 1941.
- BIANCHI C, GOLDANICH I I & ZANASI R. Contributo clinico e anatomico-patologico alla conoscenza dell'osteochondrosi dissecante — *Chir Organi Mov* 41 460 1955.
- BOBCECHKO W P & HARRIS W R. The radiographic density of avascular bone — *J Bone Joint Surg (Brit)* 42 B 6 6 1960.
- BOLNER F. Klinische und pathologisch anatomische Beiträge zur Lehre von den Gelenkknorpeln — *Deutsch J Chir* 70 363 1903.
- BRASHEAR H B Jr. Epiphyseal fractures. A microscopic study of the healing process in rats — *J Bone Joint Surg (Amer)* 41 1 1055 1959.
- BRIZZI L. Contributo sperimentale allo studio della cicatrizzazione dell'articolazione — *Sperimentale* 109 115 1953.
- BROMAN I & HÄGGQVIST C. I artiklar i Histologi — Svenska Bokförlaget Stockholm 1945.

- BROOKES M & HARRISON R G The vascularization of the rabbit femur and tibiafibula — *J Anat* 91 61 1957
- BUCHER U Befunde nach experimentellen Gelenkknorpeldefekten beim Meerschweinchen — *Schweiz Z Allg Path* 18 185 1955
- BURKHARDT L Perthes Osteochondritis dissecans und Coxa vara infantum im Tierexperiment — *Helv Chir Acta* 15 3 1948
- BYWATERS G L The metabolism of joint tissues — *J Path Bact* 44 47 1937
- CAFFEY J MADILL S H ROYER C & MORALES I Ossification of the distal femoral epiphysis — *J Bone Joint Surg (Amer)* 40 1 617 1958
- CARLSON H Reactions of rabbit patellar cartilage following operative defects. A morphological and autoradiographic study — *Acta Orthop Scand* suppl 28 1957
- COLVIN A R The clinical course and pathology of an obscure osteitis causing loose bodies in joints — *Monneosta Med* 3 65 1920
- CORNIL A & COLDRAZ P Des corps étrangers articulaires et en particulier des corps traumatiques au point de vue expérimental et histologique — *Rev Chir Orthop* 31 4 1 1905
- CRAIGMYLE M B Antigenicity and survival of cartilage homografts — *Nature* 182 1248 1958
- CROCK H V Personal communication
- DANIS A & LA BARRE J Étude de l'ossification dans les greffes de fibrocartilage fracturaire — *C R Soc Biol (Par)* 151 1386 1957
- DE PALMA A F Disease of the Knee Lippincott 1951 p 466—473
- The lesions of the knee in childhood. Osteochondritis dissecans — *Federal Clin N Amer* 2 1035 1955
- Experimental transplant of femoral osteochondral — *J Bone Joint Surg (Amer)* 42 1 186 1960
- EHRENBORG G ENCFELDT B & OLSON S I On the aetiology of the Good-Schlatter lesion. An experimental study in dogs — *Acta Chir Scand* 122 445 1961
- FAIRBANK H A T Osteochondritis dissecans — *Brit J Surg* 21 67 1953
- FEHNER A G T A study of loose bodies composed of cartilage or of cartilage and bone occurring in joints. With special reference to their pathology and etiology — *Brit J Surg* 8 432 1951
- A contribution to the pathology and etiology of osteoarthritis. With observations upon the principles underlying its surgical treatment — *Brit J Surg* 10 1 1952
- FREIBERG A H Osteochondritis dissecans — *J Bone Joint Surg* 17 1953
- GAMBIER R & VENTURINI G Ricerche sperimentali sul comportamento di innesti ossei di vario tipo nel cavo articolare — *Acta Chir Ital* 15 1955
- GIBSON A Hyaline cartilage. Degeneration and regeneration — *Canad Med Ass J* 73 44 1955
- GIES TH Histologische und experimentelle Studien über Gelenkkrankheiten

- III Zur Genese der freien chondromatösen Gelenkkörper — Deutsch Z Chir 16 330 1882
- Histologische und experimentelle Studien über Gelenkkrankheiten IV Über Heilung von Knorpelwunden — Deutsch Z Chir 18 8 1883
- HAINES R W Cartilage canals — J Anat 68 45 1933
- HAM A W Histology 3rd ed Pitman Medical Publishing Co Ltd London 1937
- HEIKEL H V A On ossification and growth of cartilaginous bones of the rabbit with a comparison of the skeletal type in the rabbit and in man — Acta Orthop Scand 29 171 1960
- HESL A Reactions of mammalian fetal tissues to injury IV Cartilage — Anat Rec 121 503 1955
- HILDEBRAND Experimenteller Beitrag zur Lehre von den freien Gelenkkörpern — Deutsch Z Chir 42 292 1815
- HIRSCH C Artroplastique cartilagineuse de la hanche — Acta Orthop Scand 21 133 1951
- HOLMDAHL D F & INGELMARK B E The contact between the articular cartilage and the medullary cavities of the bone — Acta Orthop Scand 29 156 1960
- HUNNELL D J The vascularization of cartilage — J Anat 69 47 1931
- INGELMARK B E & SAAKS J Über die Ernährung des Gelenkknorpels und die Bildung der Gelenkflüssigkeit unter verschiedenen funktionellen Verhältnissen — Acta Orthop Scand 17 303 1948
- KAPPEL M Über eigenartige Knorpelverletzungen am Caputulum humeri und deren Beziehungen zur Entstehung der freien Ellenbogengelenkkörper — Deutsch Z Chir 112 18 1917
- Osteochondritis dissecans und traumatische Gelenkmäuse — Deutsch Z Chir 127 187 1926
- KING D Osteochondritis dissecans A clinical study of 71 cases — J Bone Joint Surg 14 135 193
- KIRSCHNER M Ein Beitrag zur Entstehung der Gelenkmäuse im Kniegelenk — Bruns Beitr Klin Chir 54 417 1901
- KROH F Klinische und histologische Beiträge zur Lehre von der Osteochondritis dissecans des Kniegelenkes — Lingeneck Arch Klin Chir 149 171 1928
- LACROIX P L'ostéochondrite dissequante — Rev Belg Sc Med 13 78 1941
- Osteolyse et induction — Bull Acad Roy Med Belg 24 638 1929
- LACROIX P, FÉRALZ C & CAMIER C Contribution à l'étude histopathologique de l'ostéochondrite dissequante — Ann Anat Path (Par) 6 293 1961
- LAWRENCE J W The reactions of injured human articular cartilage J Bone Joint Surg (Brit) 39 B 548 1957
- LAWRENCE KELL A Can osteochondritis dissecans arise as a sequel of cartilage fracture in early childhood? — Acta Chir Scand 109 46 1955
- Unpublished observations

- LEB A Die Aetiologie der sogenannten Osteochondritis dissecans (König) (Eine klinische und röntgendiagnostische Studie) — Langenbeck Arch Klin Chir 131 425 1924
- LEHMANN J C Die konstitutionell schwache Epiphyse und ihre Beziehungen zur Rachitis Osteochondritis und Arthritis deformans — Deutsch Z Chir 178 11 1923
- Ist eine Wiedereinheilung osteochondritischer Gelenkmause möglich? — Deutsch Z Chir 192 88 1925
- LINDEMANN K Handbuch der Orthopädie Georg Thieme Verlag Stuttgart 1927, p 178—180
- MARTENS M Zur Kenntnis der Gelenkkörper — Deutsch Z Chir 53 318 1899
- MAXIMOW A A & BLOOM W A Textbook of Histology W B Saunders Company Philadelphia and London 1928
- MOORE J R Cartilaginous cup arthroplasty in ununited fractures of the neck of the femur — J Bone Joint Surg (Amer) 30 A 313 1948
- MORGAN J D Personal communication
- NAGURA S Das Wesen und die Entstehung der Osteochondritis dissecans Königs (bzw der Perthes Kohler II und der ähnlichen Krankheiten und Veränderungen an wachsenden Knochenenden) — Zbl Chir 61 2049 1937
- NAGURA S & KOSUGE S Die Pathogenese und das Wesen der Pertheschen Krankheit (Experimentelle Beiträge zur Entstehung der Pertheschen Krankheit) — Langenbeck Arch Klin Chir 191 347 1938
- NEUMANN W & SUTLER A Beitrag zur Frage der Osteochondritis dissecans — Deutsch Z Chir 146 219 1918
- NOVOTNY H Preventive and conservative treatment of osteochondritis dissecans — Acta Orthop Scand 21 10 1951
- Osteochondrosis dissecans in two brothers The pre and developed state — Acta Radiol (Stockh) 57 193 1952
- PREMISTER D B The cause of and changes in loose bodies arising from the articular surface of the joint — J Bone Joint Surg 6 278 1914
- FICK, M P Familial osteochondritis dissecans — J Bone Joint Surg (Brit) 34 B 147 1955
- RAO K V S An experimental study of regeneration in cartilage — J Path Bact 67 455 1954
- REHBEIN I Die Entstehung der Osteochondritis dissecans — Langenbeck Arch Klin Chir 26, 18 1910
- RIBBING S Hereditäre multiple Epiphysenstörungen und Osteochondritis dissecans — Acta Radiol (Stockh) 4 337 1954
- RIMANN H Experimenteller Beitrag zur Lehre von der Entstehung der echten freien Gelenkkörper — Virchow Arch Path Anat 180 116 1905
- RINE I A Ossification and growth of the distal ulnar epiphysis of the rabbit — J Anat 85 457 1955
- ROCHE A F & SLUNDERLAND S Multiple ossification centres in the epiphyses









THE PHANTOM PHENOMENON  
IN AMPUTATED FINNISH WAR VETERANS

BY

KAUKO A. SOLONEN

3/11/52  
BRARY  
247

MUNKSGAARD  
COPENHAGEN 1952









THE PHANTOM PHENOMENON  
IN AMPUTATED FINNISH WAR VETERANS



ACTA ORTHOPAEDICA SCANDINAVICA

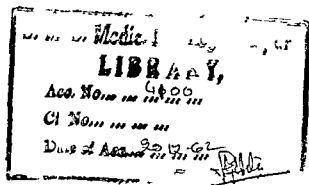
SUPPLEMENTUM NO 54

FROM THE STATE ACCIDENT OFFICE AND KASKISAARI REHABILITATION  
INSTITUTE HELSINKI

THE PHANTOM PHENOMENON  
IN AMPUTATED FINNISH WAR VETERANS

BY

KAUKO A. SOLONEN



MUNKSGAARD  
COPENHAGEN 1962





## INTRODUCTION AND REVIEW OF LITERATURE

It is a known fact that the majority of amputees after the loss of a limb or a major part of it retain a more or less distinct impression of either complete or partial existence of the lost member. Some feel this phantom phenomenon throughout their lives while for others it weakens or is otherwise modified and in some cases disappears. It almost seems as if the phantom phenomenon of an amputee were something natural though a sensation of a non-existing thing is usually considered unnatural. Such sensations have been claimed to be illusions or even hallucinations. When a phantom sensation only comprises a part of the lost limb it does not follow the anatomical areas of the nerves of the limb in question. It is regularly found that the distal most innervated and differentiated parts of the limb those that receive the greatest amount of sensory experience from the world outside are felt most clearly. Many amputees feel that they can move the phantom or its parts. Attempts have even been made to utilize the movements of a phantom limb for guiding the movements of an upper limb prosthesis at will (SIEHLOW 1951). Various factors e.g. use of prosthesis and treatment of the stump can affect the phantom size and other properties. The phantom may be painless for some even pleasant or extremely painful. The pain may differ in degree at different times and under the influence of different factors. Some believe that phantom pain only represents a more intense degree of phantom sensation (FEINSTEIN & *al* 1954) which probably results from disturbed function (WEISS 1956 WULLENWEBER 1958).

Amputees often suffer some pain in the stump. Even if the pain produced by acute or chronic trauma is disregarded pain can

usually be found in perfectly blameless amputation stumps too. These pains have many causative factors but thorough investigation often discloses that no pain arising from the stump itself is involved and the complaint must be referred to a phantom. Most of the amputees can distinguish stump pain from phantom pain but not all.

The phantom phenomenon has been studied a great deal. Early phantom studies are few which is no wonder since before the era of asepsis most amputees died soon after the operation. According to the literature (WLISS 1956 and others) however Ambroise Pare mentioned the phantom phenomenon as long ago as 1511. WEIR MITCHELL (1872) obtained his investigation material from the numerous amputees of the American Civil War. In the last decades there has been abundant material to study everywhere and so the literature dealing with the phantom phenomenon is now extensive. Nevertheless great disunity prevails as to the pathogenesis of the phenomenon.

In Finland the phantom phenomenon has been studied at least by JALAVISTO (1942, 1946, 1950 *etc.*), LILJOLIN (1946), KALLIO (1949, 1950), CEDERCRUTZ (1954, 1961) and WALLGREN (1954).

The tendency has been to explain the etiology of the phantom phenomenon in mainly two ways. The supporters of the so-called central theory (e.g. HEAD 1920, SCHILDER 1920, RIDDOCH 1941, JALAVISTO 1942, HABER 1956, WULLNWEBER 1958) believe that the phantom is there because a body image formed and fixed in the person's sensorium in the course of years remains intact despite mutilation of the body. The amputation only as it were lays bare this immaterial shape built up by sensations and engrams from the covering material part of the body. This body scheme is complete by the age of 5—7 years (BAILLY & MORISCH 1941, BROWDER & GALLAGHER 1948, RILSI & BRUCK 1950) and phantoms are only present in persons amputated after that age.

The supporters of the peripheral theory (MITCHELL 1872, PITRIS 1897, FOERSTER 1931, IRVINGSTON 1944, WHITE 1944, FALCONER & *al.* 1946, 1953) claim that the pathological sensations

of the stump e.g. irritation of a neuroma are responsible for phantom. The peripheral theory is supported by the observation that an amputee on whom a Krukenberg's lineplastic amputation has been performed feels his phantom limb split (KALLIO 1949). The peripheral theory is strongly contradicted by the fact that the parts of limbs that the phenomenon affects do not follow the peripheral nerve distribution and the phantom is not an anatomical copy of the missing part of the body by the fact that it is there immediately after amputation before neuromas have developed and that it persists even though all known afferent tracks are severed.

There are also those who believe in a purely psychogenic origin (HEAD & HOLMES 1911 BAILEY & MOERSCH 1941 PFILUGGE 1943 RANDALL & *al* 1945 KOLB 1950 1952). It is not at all difficult to describe emotional disorders of the amputees in support of the theory but there is also good reason to believe that a phantom pain having persisted long and severely is their cause and not result (LIVINGSTON 1944 FALCOVER 1953). Closest to truth is perhaps the opinion held e.g. by FEINSTEIN & *al* on the etiology of the phantom phenomenon. »Demonstrable pathological conditions in the stump in the conducting system between the stump and brain in the brain and in consciousness should be considered as different parts or aspects of one continuum.» This opinion seems to be supported also by LUNN (1948) BORS (1951) and SOLCKS (1951).

In a study published in 1955 INMAN and EBERHART found that 80 per cent of the amputees were »substantially free of pain» while some 10 per cent had »incapacitating pain» which prevented successful rehabilitation (these figures cover both stump and phantom pain). CANTY (1958) again found that over 50 per cent of amputees experience stump pain or discomfort sufficient to discontinue temporarily the use of their prosthesis» while 15 per cent had »major pain problems» because of the prosthesis for reasons arising from the stump or owing to the phantom. Although all the amputees in Canty's series had the phantom sensation immediately after amputation it did weaken in 1—2 years

and serious incapacitating phantom pain («of a major degree») was only present in some 2 per cent. LIVINGSTON'S series included less than 10 per cent of amputees with severe, permanent phantom pain. EWALT & *al* (1947) and HENDERSON & SMYTH (1948) put the figure at less than 1 per cent. According to RIDDOCH 50 per cent had a more or less painful phantom, while for PITRELS' series the figure was 97 per cent and in LERICHE'S (1937) 98 per cent.

## THE PROBLEMS

The present paper can hardly throw any additional light on the etiology of the phantom phenomenon nor on the many problems of its treatment (The lack of physiological and psychological expertise is doubtless a great weakness in making a study of this kind) It is obvious that the phantom phenomenon can only be further clarified by experimental investigations that are difficult to realise. However the series studied was so extensive and so homogenous that it may produce replies to the following questions

What is the incidence of phantom phenomenon?

What is the relationship between the phantom phenomenon and the condition of the stump

What is the phantom phenomenon like in this series of war disabled?

How does the phantom change in the course of years

How do the use of a prosthesis and activity of the stump affect the phantom?

What is the practical importance of the phantom to the amputee

What results have been achieved from treatment of phantom pain?

How can the replies to the above questions be utilized in amputation surgery in the after-care of amputees and in the production of prostheses

and serious incapacitating phantom pain («of a major degree») was only present in some 2 per cent. Livingston's series included less than 10 per cent of amputees with severe permanent phantom pain. EWALT & *al* (1947) and HENDERSON & SMYTH (1948) put the figure at less than 1 per cent. According to RIDDOCH, 50 per cent had a more or less painful phantom while for PITRES' series the figure was 97 per cent and in LERICHE's (1937) 98 per cent.

## METHOD

The data required were compiled from replies to a detailed questionnaire of 91 questions circulated to some 4 000 amputated war casualties. The questions were formulated to overlap to some extent and so an incomplete reply usually was complemented by the reply to another question. For the present study the data contained in the first 1 000 complete but otherwise unselected replies were employed. Furthermore 300 amputees were seen in person and it was found that the replies to the questionnaire on this matter of subjective observations were highly satisfactory. For the sake of uniformity the data given in the following were assembled from the written replies except in the few cases when for the sake of clarity a point had to be analysed in greater detail. These cases will be indicated.

## RESULTS

### Incidence of phantom

The phantom was felt after amputation by 945 (94.5 per cent) Forty (4 per cent) remembered that they had not felt the phantom until 6—12 months after amputation 15 (1.5 per cent) until over 2 years and 3 of them not until over 10 years had elapsed. It is obvious that these reports can only be taken to show the trend since the long lapse of time doubtless led to mistakes.

At the time of the study 847 (84.7 per cent) had a phantom which was felt in different ways and degrees

*153 (15.3 per cent) were phantom free*

### Stumps

Before trying to gain a more detailed idea of the phantom world of the subjects it is necessary to find out about the type and condition of their amputation stumps. One of the weaknesses of the present study is that it was impossible to make a personal examination of all the stumps. Whoever has had a lot to do with amputees will know, however, that the good or poor condition of the stump is best assessed by the amputee himself, especially if he already has an experience of 12—38 years. Many a stump that may look blameless is poor because of pain and tenderness while many miserable looking stumps subjectively and in practice may be good.



## Stump pain

150 amputees (15.0 per cent) reported that no significant pain<sup>1)</sup> or tenderness was present in the stump

TABLE 2. *Per cental distribution of the intensity of pain and or tenderness of stump*

| Severe | Slight | Varying | Absent | Total |
|--------|--------|---------|--------|-------|
| 18     | 60     | 7       | 15     | 100   |

This stump discomfort often in an anatomically poor stump prevented or considerably restricted the use of a prosthesis in 13 per cent of the series. In the different amputee groups sufferers from stump pain or tenderness ranged from 81 to 100 per cent. At its worst the discomfort was highly significant for instance 24 per cent of the above elbow amputees gave it as the reason why they never wore a prosthesis. Incidentally the total series included 7 (0.7 per cent) amputees who for some other reason e.g. because of a too short stump the little help the prosthesis gave or habitual attitude never wore a prosthesis.

All the amputees who reported tenderness of the stump claimed that it was constant.

Stump pain occurred as shown in the table below

TABLE 3. *Pain in the stump (per cent of total material)*

| always | often | seldom | total |
|--------|-------|--------|-------|
| 17     | 21    | 4      | 42    |

The intensity of the pain is illustrated by the fact that the pain often disturbed sleep and occasionally prevented it for 50 per cent and made work difficult for 34 per cent of those suffering from stump pain.

<sup>1)</sup> Here and in the following the word pain will be used although the amputees described their pain by a vast variety of expressions (burning, shooting, crushing, stabbing, aching, tearing, etc.).



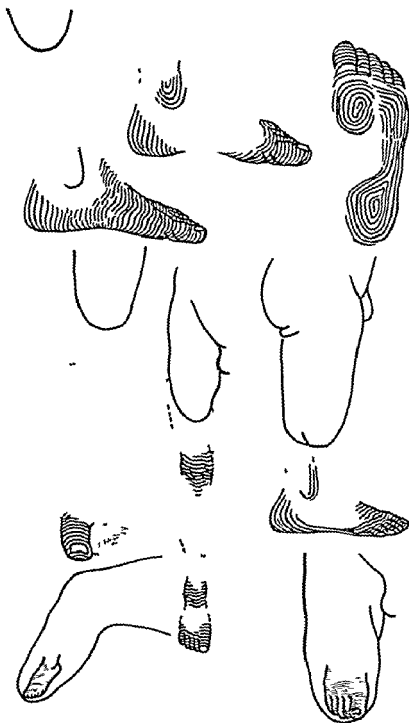
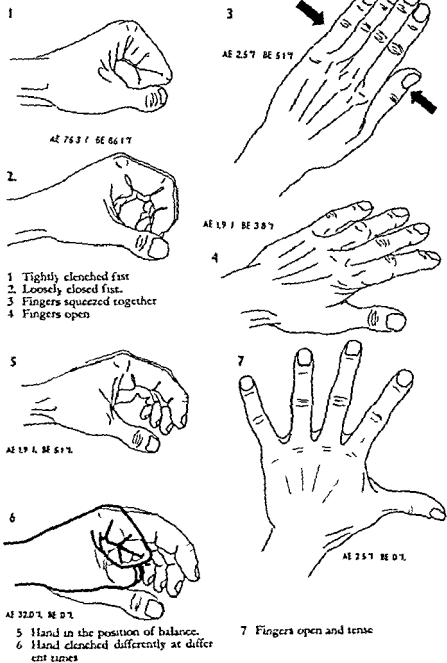
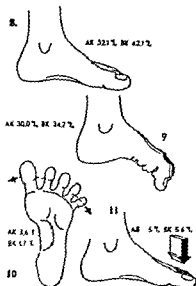
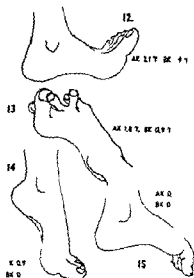


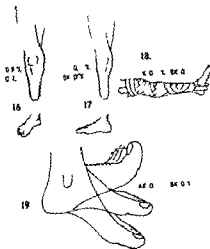
TABLE 7 *Position of the phantom*



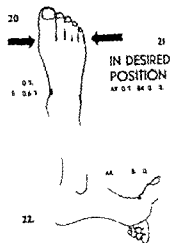
- 8 Foot in natural position  
 9 Foot and toes in plantar flexion  
 10 Toes spread and tense  
 11 Toes stretched and pressed against some lower support.



- 12 Toes in strong dorsal flexion  
 13 Toes intertwined  
 14 Foot in a rigid equinus position  
 15 The distal part of foot and toes hanging slack



- 16 Foot turned sideways  
 17 Foot strongly twisted sideways  
 18 Limb in bandage as if after first aid  
 19 Position of foot and toes varies at different times



- 20 Toes squeezed together  
 21 Foot in desired position  
 22 Big toe in extreme dorsal flexion other toes in rigid plantar flexion

## Position of the phantom limb

is illustrated by Table 7 (It should be noted that these pictures do not attempt to indicate the extent of the phantom but only its usual position. Phantom movements have not been studied.)

Similar sensations were described by the double amputees. Their phantoms are usually not symmetrical even if the amputations seem to be so.

## Phantom pain

The phantom was painless for 20 per cent of the 847 phantom positive and painful to varying degree and in different ways for 80 per cent. None of those who answered the questionnaire in writing or those who were examined in person reported the phantom sensation to be irritating if it was painless. On the other hand, no one found the phantom sensation pleasant.

## Type of phantom pain

Amputees described their phantom pain in the most varying ways. Quite often it was felt like torture performed by means of various implements of everyday life. Some of the commonest instances are assembled in Fig. 2. Phantom pain differs in many ways from other pain sensations. It often ends if the patient's mind is occupied elsewhere. The statement that phantom pain is so severe that one must go to sleep in the middle of work sounds extraordinary — the man falls asleep readily and the pain disappears. Yet the same man claimed that his phantom pain disturbed his night's sleep. One amputee reported laughing how at that very moment he suffered from 'a hell of a phantom pain'.

## Incapacitating phantom pain

The incidence of severe persistently incapacitating phantom pain was as follows:

TABLE B *Incapacitating phantom pain occurred*

|      |                         |        |          |
|------|-------------------------|--------|----------|
| In   | 3 per cent of the total | AE     | amputees |
| » 1  | » » » »                 | BE     |          |
| » 3  | » » » »                 | AK     |          |
| » 2  | » » » »                 | BK     | »        |
| » 16 | » » » »                 | Double |          |

Just under 3 per cent of all the amputees suffered from persistent incapacitating phantom pain. For a smaller series INMAN and EBERHART gave the percentages as 4. CANTY 2. EWALT & *al* HENDERSON and others below 1 per cent.

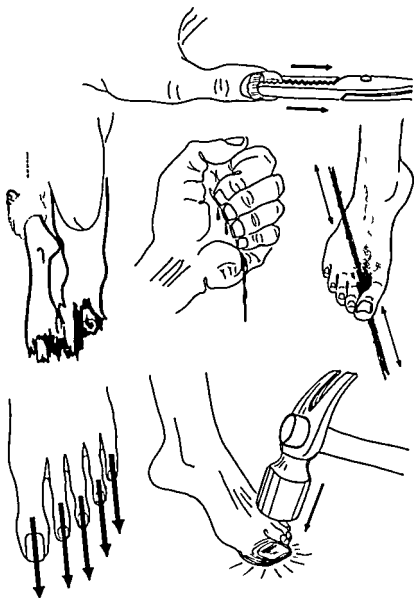
It is natural that the incapacitating pain should be a highly subjective sensation. Discomfort that totally incapacitates one person may be easily tolerated by another. For the present study phantom pain was classified as incapacitating (severe disabling pain) if it disturbed the amputee day and night and persistently prevented him from working or getting a night's rest. All those referred to this group were compelled to take drugs regularly for their pain. A large number had because of the pain submitted to surgery of the stump and nerves, spinal cord or brain, hypnosis and other therapeutical methods — all to no avail.

### The factors provoking phantom pain

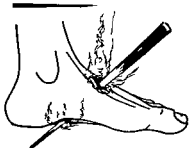
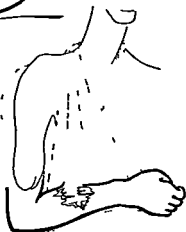
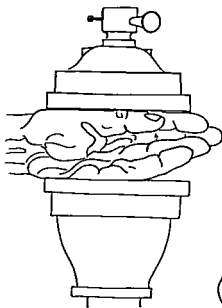
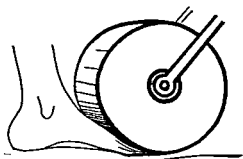
are most varied. The following is a list of the commonest in their order of incidence.

|   |           |
|---|-----------|
| Strain on the stump and the whole person          | 185 cases |
| Touching the so-called trigger point of the stump | 184       |
| Weather changes                                   | 158       |
| Poor fit of prosthesis                            | 58        |
| Coldness  | 57 »      |
| Thinking of the lost limb or phantom limb         | 26        |
| Removal of prosthesis                             | 19        |
| Moving the phantom                                | 18 »      |
| Moving the stump                                  | 10        |
| Depression  | 7 »       |

*Fig 2 Commonest descriptions of the phantom pain*







Causes which were less frequent but present in more cases than one were:

Pain in or injury to the stump fever during some illness other illness or poor condition perspiration sitting rest nervousness tension or excitement, fatigue the sauna defecation vibration wearing of prosthesis hangover draught unpleasant things (e.g. seeing somebody else's wound loneliness bad temper heat getting wet walking quickly) beer awkward posture itching sudden changes in conditions light stroking of stump massaging the stump etc.)

In many cases different causes were in play at the same time

### Factors eliminating phantom pain

Just as there were many provoking factors there was a great variety of means to stop the pain. But their effect was always transient: no treatment proper is in question.

The most popular methods were

- Holding the stump upright
- Hanging the stump
- Complete rest of the stump
- Movements of the stump
- Walking vigorously
- Walking slowly
- Swimming
- Shaking the stump
- Swinging the stump
- Slow movements of the stump
- Cold water
- Hot water
- Warm water
- Alternate hot and cold baths
- Massaging the stump
- Knocking at the stump
- Beating the stump numb
- Pressing the stump
- Pressing on the trigger point
- Distracting the thoughts
- Concentrating on the phantom
- Dry heat
- Sauna
- Tobacco

Drugs  
 Alcohol  
 Hot sand  
 Spirits rub

Furthermore coffee sense of humour strong attitude against pain cursing use of prosthesis removal of prosthesis complete rest hard work coitus urination (AK) gaiety nice weather rowing (AE) etc

The above lists show that for different persons the provoking and the eliminating factors are often the same. Many definitely effective factors seem completely ineffective everyday happenings *others again have a pronounced effect either on the organism or the psyche*. There is also persistent phantom pain apparently not provoked by any external factor or psychic function. Nor has it always been possible to find any lay relief for the worst pain.

### Development of phantom and phantom pain in the course of years

It is obvious that the phantoms which are now felt as shortened originally were the length of the lost limb. It has not been possible afterwards to time their gradual shortening. Otherwise too the dates of the changes in the phantom are so difficult to establish — unless connected with some therapeutical measure — that they have not been taken into account here. The present work was limited mainly to studying the changes in the intensity of phantom pain.

The distinctness of the phantom and phantom pain was reduced between amputation and the date of examination as follows

TABLE 9 *Phantom pain relieved*

| AE amputees | 9 per cent of the phantom positive |   |   |   |
|-------------|------------------------------------|---|---|---|
| BE    »     | 18                                 | » | » | » |
| AK    »     | 17                                 | » | » | » |
| BK    »     | 12                                 | » | » | » |
| F      »    | 1                                  | » | » | » |

Phantom and phantom pain gained in intensity as follows

TABLE 10 *Phantom pain aggravated*

|             |                                    |
|-------------|------------------------------------|
| AE amputees | 4 per cent of the phantom positive |
| BE »        | 6 » » » » »                        |
| AK »        | 9 » » » » »                        |
| BK »        | 7 » » » » »                        |
| Double »    | 6 » » » » »                        |

A total of 15 amputees (1.5 per cent) reported that the phantom disappeared spontaneously 1 week — 13 years after amputation. Very many reported that the occurrence of the phantom had become less and less frequent but there are instances of the opposite too.

### Influence of prosthesis wearing on the phantom

About 35 per cent of the amputees reported that the wearing of a prosthesis affected the phantom sensation. The effects of a faultless prosthesis only are discussed here. Table 11 lists the principal observations.

TABLE 11 *Influence of prosthesis wearing on the phantom*

|   |             |
|---|-------------|
| Phantom is lengthened to equal the prosthesis or less | 9 per cent  |
| Phantom is merged into the prosthesis                 | 22 » »      |
| Phantom is shortened <sup>1)</sup>                    | below 1 » » |
| Phantom and phantom pain disappear                    | below 1 » » |
| Phantom pain is aggravated                            | 0.5 » »     |
| Amputee reports several effects <sup>2)</sup>         | 1.5 » »     |

Amputees sometimes exploit the phenomena described above especially where the wearing of a prosthesis either removes or reduces the phantom or phantom pain. Some amputees may by

<sup>1)</sup> This group includes e.g. a foot amputee whose foot phantom is shortened as if squeezed together but the toes remain whole.

<sup>2)</sup> A foot amputee feels that sometimes «a heavy shoe pressed the phantom foot exhaustingly» that is to say the strain on the stump is felt as such in the phantom limb.

this means secure their night's sleep. Others again believe that the wearing of a prosthesis is more natural when his «own limb» the phantom is lengthened to equal the prosthesis or merges with it.

### Results of treatment of phantom pain

In order to eliminate the high potential error the data for this part of the study were collected only from the 300 amputees seen in person. Nevertheless there were some patients who could not say for certain whether the treatment was given mainly for phantom pain or for stump discomfort. In any case the effect of treatment on the phantom is recorded here. The following table lists the treatments and the results. Methods of physiatric treatment are not included as the patients are least able to provide reliable information on them.

The treatments listed therefore produced permanent and complete relief (after a follow up period of 1—18 years) for 8 per cent, permanent relief for 29 per cent, transient relief for 11 per cent.

TABLE 12 *Effect of certain treatments on phantom pain*

|                                 | Phantom pain disappeared for good | Phantom pain was relieved | Phantom pain disappeared or was relieved temporarily | No effect | Phantom pain was aggravated | Cases |
|---------------------------------|-----------------------------------|---------------------------|--|-----------|-----------------------------|-------|
| Sympathectomy                   | 2                                 | 2                         | 1  | 9         | 1                           | 15    |
| Revision of stump <sup>1)</sup> | 2 <sup>1)</sup>                   | 9                         | 2  | 13        | 1                           | 27    |
| Excision of neuroma             | —                                 | 6                         | 5  | 8         | 2                           | 21    |
| Reamputation                    | 2                                 | 4                         | —  | 3         | —                           | 9     |
| Laboromy                        | —                                 | —                         | —  | 1         | —                           | 1     |
| Hypnosis                        | —                                 | 1                         | —  | 2         | —                           | 3     |
| Total                           | 6                                 | 22                        | 8  | 36        | 4                           | 76    |

<sup>1)</sup> Only stump revisions made a minimum of 2 years after primary amputation are included.

<sup>2)</sup> In both cases an excision of the fibular stump was involved.

no effect at all for 47 per cent and aggravation for 5 per cent of the patients. The most effective treatment seems to have been reamputation which in fact includes the treatments that were next most effective, viz revision and removal of neuroma. The effect of sympathectomy too is worth noting.

However, the old observation is confirmed that none of these treatments has any specific effect on phantom pain and that phantom pain or the phantom phenomenon is not a functional disorder of a given precisely delineated area of the nervous system.

### Effect of post-operative rehabilitation on the amputees phantom phenomenon

The writer holds the opinion shared by e.g. BOSHAMMER, WEISS and WULLENWEBER that early post operative rehabilitation has the most favourable effect in relegating the phantom phenomenon to a secondary position in the amputee's life. This point cannot however be studied from the present series as the lapse of time since the amputation is too long. The subjects as a rule received their prostheses 6—24 months after amputation, most of them obviously unnecessarily late as a result of war time difficulties. The standard of prostheses in our country at that time was not very high. From the point of view of post operative rehabilitation and prosthetic service the treatment of our amputated war veterans must be considered unsatisfactory in the main. On the other hand it is remarkable that the phantom discomforts reported on above are not essentially more numerous or severe than those presented in some statistics compiled from apparently better conditions (cf. above).

## DISCUSSION AND CONCLUSIONS

The phantom phenomenon which probably must be equated in the first place with a complication of the amputation occurred in some 85 per cent of the amputees of the present series examined an average of 14 years after amputation and 80 per cent of them reported a painful phantom. Since the phantom pain was so severe that it was disabling in nearly 3 per cent of the patients the complication is worth remembering for those admittedly rare cases with no vital indications for amputation.

A finding of interest to amputation surgery is that with good stumps the phantom phenomenon is usually less severe than with poor stumps.

The ways in which the phantom phenomenon occurred and the other characteristics of phantom sensation are very much the same as those described by other writers.

The phantom phenomenon and phantom pain generally change in the course of the years. The change is for the good i.e. relief of sensation and pain about two or three times more often than for the worse.

Wearing an artificial limb affected the phantom of over one third of the amputees and the effect was usually either void of subjective significance or in one way or another positive to the amputee. There might be some justification for believing that this effect of wearing a prosthesis is the joint result of the artificial limb as such and the accompanying activation of the stump towards the original function of the lost limb.

The practical importance of the phantom phenomenon to the amputee must be considered negative. Every amputee without a

phantom is definitely better off than his phantom positive companion in misfortune

The percentages quoted above for phantom pain are weighty arguments for the negative practical significance of the phantom phenomenon. The figures gain significance from the knowledge of how little phantom pain can be affected and how much the success or aggravation produced by therapeutic attempts is still haphazard today. It is essential however to try and analyse the factors provoking phantom pain in each individual case although some of these factors are hard to escape. Nevertheless the precise and skilful making and fitting of the prosthesis for instance is of great value. Parallely with and as an integral part of good prosthetic service every amputee should be rehabilitated to active and useful life with as little delay as possible.



## SUMMARY

A questionnaire on the phantom phenomenon was answered in writing by 1 000 amputated war veterans and 300 were examined in person

The lapse of time from the amputation ranged from 12—38 years At the time of the examination 15 per cent were without a phantom In 85 per cent the phantom occurred in the most varying ways

About 70 per cent of the amputees with a good stump had a phantom and for those with a poor stump the percentage was about 87 Some 61 per cent of the phantom negative had stump discomfort

The phantom of 20 per cent was painless of 80 per cent painful None considered the phantom phenomenon agreeable

About 3 per cent of the amputees had a persistent incapacitating phantom pain resistant to treatment

The phantom pain was most varying in type and intensity and the factors provoking and relieving it were numerous

About 5 per cent of the amputees felt their phantom in the size and shape of the whole lost limb others felt only parts of the lost limb usually considerably shortened

The phantom sensation of some 44 per cent was continuous others had it less frequently

The incidence of phantom was higher after proximal than distal amputations The double amputees felt the phantom for both lost limbs in every case studied

In the course of time the phantom tended to be relieved 2—3 times as often as it tended to be aggravated

The wearing of a prosthesis affected the phantom directly in about 35 per cent of the phantom positive cases and the effect was mostly insignificant sometimes favourable

The results of therapy for the phantom pain in the present series too were on the whole unsatisfactory, and the effect of treatment seemed mostly haphazard

The incidence of the phantom phenomenon and phantom pain and the degree of severity of the phantom pain are of such a magnitude that they must be borne in mind in considering and performing an amputation and when the amputee is rehabilitated

### Acknowledgement

The writer wishes to express his best thanks to the Disabled Ex-Servicemen's Association for help in assembling the material and for having the publication translated into English and printed at their expense

## REFERENCES

- BAILEY A A and MOERSCH F P The phantom limb — *Canad M A J* 1941 45 37
- BORS E Phantom limbs of patients with spinal cord injury — *Arch Neurol and Psychiat* 1951 66 610
- BOHAMMER Acc to Wullenroether
- BROWDER J and GALLAGHER J P Dorsal chordotomy for painful phantom limb — *Ann Surg* 1948 128 456
- CANNY T J Amputation stump pain — *U S Armed Forces M J* 1958 9 635
- CEDERCREUTZ C Hypnotic treatment of phantom sensations in 100 amputees — *Acta Chir Scand* 1954 107 158
- CEDERCREUTZ C Multiple phantom — *Nord Med* 1961 66 1537
- ELLOVEN A L'effet de la sympathectomie sur le fantôme douloureux d'un amputé — *Acta Chir Scand* 1946 93 131
- EWALT J R RANDALL G C and MORRIS H The phantom limb — *Psychosom Med* 1947 9 118
- FALCONER M A and LINDSAY J S B Painful phantom limb treated by high cervical chordotomy — *Brit J Surg* 1946 33 301
- FALCONER M A Surgical treatment of intractable phantom limb pain — *Brit M J* 1953 1 299
- FEINSTEIN B LUCE J C and LANGTON J N K Chapter 4 in Human limbs and their substitutes Edited by KROSTAG P F and WILSON P D 1954 McGraw Hill New York
- FOERSTER O Über das Phantomglied — *Med Klin* 1931 27 497
- HABER W B Observations on phantom limb phenomena — *Arch Neurol Psychiat* 1956 75 624
- HEAD H and HOLMES G Sensory disturbances from cerebral lesions — *Brain* 1911 34 102
- HEAD H Studies in neurology London 1920 Acc to Lunn

- HENDERSON W. R. and SMYTH G. E. Phantom limbs — *J Neurol Neurosurg & Psychiat* 1948 2: 88
- INMAN V. T. and EBERHART H. D. The lower extremity clinical study — its background and objectives. — *Artif Limbs* 1955 2: 4
- JALAVISTO E. Oma ruumiimme havaintomme kohteena. — *Duodecim* 1942, 5: 187
- JALAVISTO E. Oman ruumiin havainnosta esineinvarianssina kokeita yläraajoja amputoiduilla. — *Ajatus* 1943 5
- JALAVISTO E. Perception of weight and the phenomenal regression to the «real» weight (thing constancy phenomenon) — *Acta Physiol Scand* 1946 11: 111
- JALAVISTO E. and SOLRÄNDER P. Observations on arm amputees — *Ann Acad. Scient Fenn A V Medica Anthropol* 1948 17: 1
- JALAVISTO E. Adaptation in the phantom limb phenomenon as influenced by the age of the amputees. — *J Gerontol* 1950 5: 339
- KALLIO K. E. Is excision of neuromas unadvisable. — *Ann Chir et Gyn Fenn.* 1949 38: 165
- KALLIO K. E. Phantom limb of forearm stump cleft by kineplastic surgery — *Acta Chir Scand* 1949 99: 121
- KALLIO K. E. Permanency of result obtained by sympathetic surgery in the treatment of phantom pain. — *Acta Orthop Scand* 1950 19: 391
- KOLB L. C. Psychiatric aspects of treatment for intractable pain in the phantom limb — *M Clin. North America* 1950 34: 1029
- KOLB L. C. Treatment of the acute painful phantom limb — *Proc Staff Meet. Mayo Clin* 1952, 27: 110
- LERICHE, R. La chirurgie de la douleur — Masson & Cie Paris 1937
- LIVINGSTON K. E. The phantom limb syndrome. A discussion of the role of major peripheral nerve neuromas. — *J Neurosurg* 1945 2: 251
- LIVINGSTON W. K. Pain mechanism, a physiologic interpretation of causalgia and its related states — The Macmillan Co 1944 New York
- LENN V. Om legemsbevistheden Diss. Ejnar Munksgaard 1948 Copenhagen
- MITCHELL, S. W. Acc. to Weiss.
- PENFIELD W. and BOLDREY E. Somatic motor and sensory representations in the cerebral cortex of man as studied by electrical stimulation. — *Brain* 1937 60: 389
- PFLÜGGE, H. Zur Entstehung des Phantomgliedes — *Deutsche Ztschr f Nervenhe* 1942—1943 154: 199
- PITRES A. Acc. to Weiss.
- RANDALL, G. C. F. WALT J. R. and BLAIR H. Psychiatric reaction to amputation. — *J A M A* 1945 123: 645

- RIESE U. and BRUCH G. Le membre fantôme chez l'enfant. — *Rev Neurol* 1930 83 221
- RIDDOCH G. Phantom limbs and body shape. — *Brain* 1941 64 157
- SCHILDER A. Das Körperschema. — Berlin 1924
- STERNOW K. Der Verlust des Arms, ein Phänomen gesteuerter Elektrizität. — *Zentralbl. f. Chir.* 1931 76 1123
- SOLCKS W. Two stage disappearance of phantom limbs. — *Wien. klin. Wochenschr.* 1951 63 329
- WALLGREN G R. Phantom experience at spinal anesthesia. — *Ann. Chir. et Gyn. Fenn.* 1934 43 486
- WEISS A A. The phantom limb. — *Ann. Int. Med.* 1936 44 668
- WHITE, J. C. Pain after amputation and its treatment. — *J. A. M. A.* 1944 124 1027
- WILLENWEBER R. Zur Pathogenese des Phantomschmerzes. — *Fortschr. d. Neurol. Psychiat.* 1958 26 495









ACTA ORTHOPAEDICA SCANDINAVICA  
Supplementum no 55

---

FROM THE UNIVERSITY CLINIC OF ORTHOPAEDICS AND TRAUMATOLOGY  
(CHIEF PROFESSOR K E KALLIO) AND THE ROENTGEN DIAGNOSTIC  
DEPARTMENT OF THE UNIVERSITY SURGICAL CLINICS  
(CHIEF DOCTOR ROLF KÖHLER) HELSINKI FINLAND

# CONTRAST EXAMINATION OF LUMBAR INTERSPINOUS LIGAMENTS

*(Ligamentographia interspinosa lumbalis)*

BY

ROLF KÖHLER



MUNKSGAARD  
Copenhagen 1962



**CONTRAST EXAMINATION OF LUMBAR  
INTERSPINOUS LIGAMENTS**



**CONTRAST EXAMINATION OF LUMBAR  
INTERSPINOUS LIGAMENTS**



ACTA ORTHOI AEDICA SCANDINAVICA

Supplementum no 25

FROM THE UNIVERSITY CLINIC OF ORTHOI AEDICS AND TRAUMATOLOGY  
(CHIEF PROFESSOR K E KALLIO) AND THE ROENTGEN DIAGNOSTIC  
DEPARTMENT OF THE UNIVERSITY SURGICAL CLINICS  
(CHIEF DOCTOR ROLF KÖHLER) HELSINKI FINLAND

# CONTRAST EXAMINATION OF LUMBAR INTERSPINOUS LIGAMENTS

*(Ligamentographia interspino-sa lumbalis)*

BY

ROLF KÖHLER

MUNKSGAARD  
Copenhagen 1962



PRINTED IN FINLAND BY TILGMANN  
HELSINKI 196



# CONTENTS

|   | Page |
|---|------|
| I Introduction  | ~    |
| II Anatomic and histopathologic survey  | 1    |
| III Object of the examination   | 11   |
| IV Series and method  | 13   |
| V General survey of the presence of ligamentous changes in the series   | 18   |
| — Age and sex   | 18   |
| — Localization  | 19   |
| - Posture of the lumbar spine   | 19   |
| Height of the spinous process   | 21   |
| The capacity of the lumbar spine for extension  |      |
| Displaced spinous processes   | 24   |
| Ligament thickness  | 25   |
| VI The different types of changes in the three lower interspinous ligaments: their incidence and roentgenologic diagnosis | 28   |
| Normal ligaments  | 8    |
| Ligaments in displaced spinous processes  | 30   |
| Cavity formation  | 32   |
| Ruptures in ligaments   | 38   |
| a) Partial ruptures   | 39   |
| b) Complete ruptures  | 43   |
| Flaccid ligaments   | 46   |
| VII Disk degeneration: trauma of the lumbar spine and changes in the interspinous ligaments                               | 49   |
| - Disk degeneration   | 51   |
| Trauma  | 52   |
| VIII Changes in the three lower lumbar interspinous ligaments and low back pain   | 54   |
| Discussion  | 59   |
| IX Summary and Conclusions  | 61   |
| Acknowledgements  | 62   |
| References  | 63   |



PRINTED IN FINLAND BY TILGMANN  
HELSINKI 1962

## I INTRODUCTION

Ligamentous changes in the two lower interspinous space were often observed by KALLIO in connection with operations for prolapsed disk. The supraspinous ligament was thin and inelastic while the interspinous ligaments appeared relaxed, displayed an irregular surface and appeared to be fat infiltrated or fibrotic, some were almost transparent and extremely thin while others were completely flaccid whatever their macroscopic appearance. In numerous cases this author observed rents stretching half way across the ligaments or total defects, rounded holes through which it was possible to pass a sound to the opposite side. The corresponding Bewegung segment showed in many cases increased mobility and the spinous processes sometimes displayed irregular contours or marginal deposits of cartilaginous or osseous consistency.

A search of the literature produced highly divergent, not to say conflicting, or totally inadequate accounts of the anatomic and function conditions of the lumbar interspinous ligaments. This was true for example of the course of the fibre bundles. Nor was any information found concerning the presence of congenital defects in these ligaments. Unequivocal facts relating to the supraspinous ligament were few.

The lower lumbar spine is of essential surgical orthopedic significance mostly because of the mixed pathology of the intervertebral disk. However KALLIO considered from his operative experience that the posterior ligaments in the lumbar spine which bind together the spinous processes merit greater clinical attention than they have hitherto received. He emphasized the necessity of detailed anatomic analysis of the supraspinous and interspinous ligaments and suggested to RISSANEN that he should investigate the anatomy and pathology of the interspinous ligaments of the lumbar spine in autopsy material. This author presented his results in 1910 as a doctoral thesis.

It was held by KALLIO that there was no satisfactory preoperative procedure for the establishment of changes in the interspinous ligaments although it might be of value in many cases. The present writer had to admit his inability to demonstrate these ligamentous changes roentgenologically. It was of no help basing the solution on the assumption that the differences

between the spinous distances in a flexed and extended spine in roentgenograms taken usually were significantly dissimilar in normal and pathological ligament. It proved necessary to resort to other expedient and it appeared that a positive contrast medium might overcome the absence of contrast differences between the ligament and the surrounding soft parts and thus enable a roentgenologic diagnosis of the ligament to be made. A particularly ingenious injection of contrast medium of this type produced a demonstration of the lumbar interspinous ligaments in the form of filling defects. A preliminary report of the investigation method applied to 100 clinical cases was published in 1939.

## II ANATOMIC AND HISTOPATHOLOGIC SURVEY

Vertebral bodies, neural arches and processes constitute the inner stable bony structure of the lumbar spine which is joined into a whole by various kinds of softer tissue. The anatomist LUSCHKA (1858) took an interest in the functional conditions in the spinal column and regarded the intervertebral disks as the Halbgelenke of the human body. The axis of rotation of the dorsoventral movements of the lumbar spine lies within the area of the small joints.

The pathology of intervertebral disks has attracted considerable and sustained interest during the last few decades. Mention should be made especially of the intensive research on the spine which flourished in Germany during the latter half of the 1920. The pathologist SCHMORL deserves mention in this respect before other investigators. He gave much new knowledge to which later workers were able to subscribe.

The study of the spine has been concerned almost exclusively with diverse conditions in the vertebral bodies and intervertebral disks and little attention has been paid to the posterior region of the lumbar spine. This applies not least to the interspinous ligaments and is somewhat surprising in view of the considerable degree of movement between the spinous process in extension and flexion which causes either a stretching or compression of the soft parts between the bony surfaces. Extreme movements would seem to contribute to producing changes in the ligaments which cannot be entirely immaterial for the function or clinical symptomatology of the spine. RISSAVALA's detailed study of a large material threw new light on several problems relating to the supraspinous and interspinous ligaments of the lumbar spine. The essential points of his results are given in what follows.

This author considered that the supraspinous ligament attached to the tips of the spinous process constituted the most effective limitation to excessive ventral flexion of the spine. He found that this ligament was contrary to earlier opinion absent in the lower parts of the lumbar spine. The dorsal and medial part of the lower interspinous ligaments assumed some of the duties of the supraspinous ligament in preventing excessive moving apart of the spinous process. The supraspinous ligament undergoes considerable changes with age the most characteristic of these being metaplasia

of the tendinous tissue into fibrocartilage, calcification and metaplastic ossification. No signs of rupture of the interpinous ligament were evident in any of the cases, even on microscopic examination.

Rissanen stated that the general course of the fibre bundles in the interpinous ligaments ran from behind downwards and forwards. Microscopically the ligament was seen to be composed chiefly of collagenous bundles with a very few elastic fibres. The ligament is thus capable of withstanding considerable traction but no notable stretching. By the twentieth year of life change began gradually to appear in the ligaments. Metaplasia of the tendinous tissue into fibrocartilage takes place and breaking of the fibre bundle, fragmentation and finally cavity formation occur in the middle of the ligament. In older subjects the cavities often ultimately become formations reminiscent of true joints.

Ruptures were established in the lower interpinous ligaments, mostly in the lower two, in a total of 21 per cent of subjects over 20 years of age. Microscopic examination in all cases demonstrated various degenerative changes, of which cyst-like degeneration was the most prominent. The degenerative changes appearing early in the interpinous ligaments and the cavity formation developing in the them, as well as the ligament rupture, were all attributed to a heavy local mechanical strain caused by anatomical factors, as well as to certain other more general conditions affecting the organism as a whole.

### III OBJECT OF THE EXAMINATION

As already mentioned briefly in the introduction the writer had elaborated a roentgenologic method of examining the lumbar interspinous ligaments and a preliminary report of the method appeared in 1939. The present study sums up the observations made and the results achieved with the aid of the method in examination of the three lower lumbar interspinous ligaments in a clinical series.

The examination was limited to the ligament mentioned since the majority and the most extensive of the lumbar interspinous changes are situated in the interspaces. This was the case even with all the ligamentous ruptures in RICHARDS' material. The restriction of the examination site was also necessitated by the area of occurrence of lumbar disk prolapse. In the present series it was invariably in the three lower intervertebral spaces.

One hundred and ten cases of the total 229 cases of the series had undergone a lumbar spine operation. The interspinous ligaments were carefully examined and the findings were recorded in the operation report. The writer himself was often present at the phase of the operation and was able to compare the preoperative roentgenologic finding with the ligamentous morbidity which was exposed. In the other cases the roentgenologic finding was compared with the operation report. Conformity with the operation findings was very good in about 30 per cent of the cases.

The roentgenologic examination failed in 3 cases to demonstrate cavities or ruptures in an interspinous ligament. Owing to the latter being imperfectly outlined by the contrast medium the subjects were either obese or had very firm dorsal muscles. Relaxed ligaments escaped detection in 4 cases; again the reason can be placed upon a faulty injection technique. As will be pointed out later the diagnosis of flaccid ligaments requires intimate contact between the contrast medium and the ligament—a desideratum that in these cases was not adequately relieved. Minor deviations from the operation findings were evident in 6 cases; these were of such a nature that it is not unlikely that the ligamentous changes were analyzed more accurately and correctly by the roentgenologic examination.

It would appear in view of the foregoing that there is some justification in claiming that a refined roentgenologic examination of the three lower lumbar

interspinous ligaments is capable of demonstrating any changes without necessarily resort to operation.

Neither of the methods approaches the exactitude obtained in Riva's systematical dissection of the ligament. On the other hand, both are satisfactorily reliable from the practical and clinical view points, which support a review of the investigation and an analysis of the results.

The present investigation was undertaken with a view to determining —

- 1) The kind of ligament change that may be diagnosed roentgenologically and their roentgenologic characteristic.
- 2) The distribution of the changes by age and sex.
- 3) The effect of posture, height of the pinous process, range of extension of the lumbar spine and displaced pinous process on the changes in the interspinous ligaments.
- 4) The relationship between the thickness and any changes in the ligament.
- 5) The origin of the change in ligament: a) in connection with degeneration and prolapse of vertebra; b) after spinal trauma.
- 6) Whether any correlation exists between the change in the lumbar interspinous ligaments and low back pain.



## IV. SERIES AND METHOD

The series consisted of 229 cases on which ligamentography was performed in the period between January 1958 and May 1960. One hundred and eighty nine cases had low back pain or sciatica while the remaining 40 had never been so afflicted, had been admitted to hospital for other reasons and had volunteered to act as controls.

The age distribution is seen in the following table.

|             | Controls |        |       | Cases with back pain |        |       |
|-------------|----------|--------|-------|----------------------|--------|-------|
|             | Mal      | Female | Total | Mal                  | Female | Total |
| 11—20 years | —        | 1      | 3     | 3                    | 3      | 6     |
| 21—30 "     | 10       | 3      | 13    | 24                   | 3      | 27    |
| 31—40 "     | 11       | 4      | 15    | 48                   | 14     | 62    |
| 41—50 "     | 3        | 3      | 8     | 41                   | 22     | 63    |
| 51—60       | 1        | 0      | 1     | 0                    | 6      | 6     |
| 61—70       | 0        | 0      | 0     | 1                    | 2      | 3     |
|             | 25       | 11     | 40    | 137                  | 48     | 185   |

*Table 1. Age distribution of the investigation series of 229 cases*

The male:female ratio in both the principal groups was almost 3:1. The youngest and the oldest age categories as well as the other groups of the control material are unusable for statistical purposes when separated. The control material on the other hand, should be usable in its entirety for a statistical analysis.

The age distribution of the two principal groups of the total series shows a distinctly lower age trend among the cases without back symptoms. This was as to be expected, degenerative changes which are the main cause of low back pain increase with advancing age. Indeed the author was surprised that it was difficult to find subjects over 40 years of age who had never felt pain referable to the lumbar spine. This was one reason for the somewhat unsatisfactorily large number of controls. The other reason was that quite a number of those approached were not inclined to submit voluntarily without any benefit to themselves to an examination involving a number of pricks with a pin and thus not completely painless.

interspinous ligaments is capable of demonstrating any change without necessarily resort to operation.

Neither of the methods approaches the exactitude obtained in the x-ray's meticulous direction of the ligament. On the other hand both are satisfactorily reliable from the practical and clinical view point which support a review of the investigation and analysis of the results.

The present investigation was undertaken with a view to determining:

1) The kind of ligament change that may be diagnosed roentgenologically and their roentgenologic characteristics

2) The distribution of the change by age and sex

3) The effect of posture, height of the spinous process, range of extension of the lumbar spine and displaced spinous process on the change in the interspinous ligament

4) The relationship between the thickness and any change in the ligaments

5) The origin of the change in ligaments a) in connection with degeneration and prolapse of intervertebral discs b) after spinal trauma

6) Whether any correlation exists between the change in the lumbar interspinous ligaments and low back pain

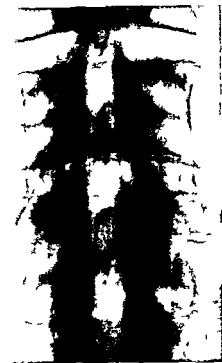


Fig. 1. Note 1 finding. Bilateral paraspinal injection of contrast medium. Interspinous ligament appear a filling defect located between the spinous process and medially sharply demarcated. Lateral contour of the contrast medium, moderate breaking.

was often streaky (Fig 2). The writer expected that the penetration of contrast medium into or through the ligament and the irregular contour of its medial border might reveal a pathologic ligament.

In the first 60 cases the contrast medium was injected unilaterally and on the side of the patient's main symptoms. It was believed that in this way a defect would be better revealed by a flow of contrast medium to the side un.injected. Bilateral injection, however, eventually proved to give a more complete result without loss of the advantage gained by the earlier method. The bilateral injection technique was consequently employed exclusively and is as follows:

The patient is placed in a lateral position on the Bucky table with the lumbar spine flexed. The three lowermost interspinous spaces are palpated and marked and the skin is cleansed. A 20 ml syringe fitted with a No 12 needle and filled with 1% xylocaine exadrine is employed for the first

The method of investigation was based on the fact that the space which lies between two adjacent pinous processes is filled completely by the interpinous ligament located in the pinous plane (i.e. sagittal plane) (shown in Fig. 1).

The ligaments in children form a continuous tendinous membrane and in the study carried out by RUSSELL (101 subjects aged 0-10 years) they were completely intact. The ligaments showed no perforation or other defect in this age group if the axial and lumbar skeleton of the child was normal. It is possible on the other hand to demonstrate a variety of changes in the lumbar pinous ligaments from the 20th year of age onward.

There is no likelihood of eliciting the changes in conventional roentgenogram, the evaluation of which requires as was pointed out in the introduction a change in the contrast condition in the interpinous region. When positive contrast medium was injected along the ligamentous wall between the pinous process the interpinous ligaments appeared in the anteroposterior roentgenograms of the lumbar pinous sharply demarcated usually slightly spindle-shaped filling defect between the contrast medium deposit which the ligament prevented from spreading right up to the median line. The contrast medium usually spread irregularly in the lateral direction and

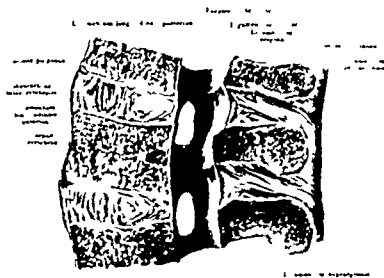


Fig. 1. Median section through the lumbar spine with the ligamentous interpinous ligament (L. interpinous) at the level of L. 5.



Fig. 1. Normal finding. Bilateral paragonitoul injection of contrast medium. Interspinous ligaments appear as filling defects located between the spinous process and medially sharply demarcated. Lateral contour of the contrast medium with moderate breakline.

was often streaky (Fig. 2). The writer expected that the penetration of contrast medium into or through the ligament and the irregular contour of its medial border might reveal a pathologic ligament.

In the first 60 cases the contrast medium was injected unilaterally and on the side of the patient's main symptoms. It was believed that in this way a defect would be best revealed by a flow of contrast medium to the side un.injected. Bilateral injection, however, eventually proved to give a more complete result without loss of the advantage gained by the earlier method. The bilateral injection technique was consequently employed exclusively and is as follows:

The patient is placed in a lateral position on the Bucky table with the lumbar spine flexed. The three lowermost interspinous spaces are palpated and marked and the skin is cleansed. A 20 ml syringe fitted with a No 12 needle and filled with 1% xylocaine exadrine is employed for the first

injections. A small quantity of the anesthetic is injected subcutaneously in the midline over the I to S4 interpinous space. The needle is then introduced to its entire length at right angle to the skin but 0.5 to 1 cm lateral to the midline in the sagittal plane along the interpinous ligament.

The needle first encounters a certain amount of resistance which after the point has penetrated a distance of about 1 cm is reduced provided the ligament has not been penetrated. Five to seven ml of the anesthetic are then injected and the procedure is repeated on the opposite side. The two lower ligaments above are similarly treated. By placing the tip of the index finger on the adjacent pinous process it is easy to locate the ligament provided the subject is not too obese. Satisfactory examination of obese person is difficult and the exact deposition of contrast medium is a basic condition for a reliable myelogram result and cannot be achieved when the tips of the processes are impossible to palpate through the covering layer of fat.

After the administration of anesthetic 1 to 2 ml of the contrast medium were injected at the first site of insertion. A test dose of this kind may be useful in checking sensitivity to the drug. Only one of the authors ever developed urticaria during the examination and none of them reacted to the test dose.

The time of injection is repeated 10 to 15 minutes later with a contrast medium substituted for the anesthetic. 1 to 2 ml of the medium being introduced in the same way as close as possible to the ligamentous wall. At first we used a 20% monomerized water soluble preparation (Centurox, Lundbeck) contrasted with iodocholyl in order to prevent injury in the event of accidental introduction into the lumbar canal. We employ the emulsions for lumbar myelography. The density of the film was fairly satisfactory but more recent methods hardly to be feared a slightly more concentrated preparation such as Iopromazin 30% seemed to be preferred. This considerably improved the quality of the film and is now used exclusively.

After the injection of the contrast medium a roentgenogram is obtained in the interepitrochlear projection with the patient supine and with the torso reduced as much as possible. All the ligament measurements given follow were obtained from this film. An exposure with the body in a ventrodorsal position is possible may also be indicated in order to know the degree to which the thin and narrow ligament may increase in thickness when the pinous processes approach one another. In pathologic conditions myelography of the lumbar plexus was performed with the patient in the same position. The complementary examination will probably demonstrate ligament

mentou detail which have been obscured in the routine roentgenogram through summation. A tomographic examination also helps in locating the pathologic ligament section in the anteroposterior direction and determining its dimension in depth.

The contrast medium is for the most part absorbed within an hour. The patient usually tolerates the examination fairly well. Repeated injections for the induction of anaesthesia, however, inconvenience sensitive individuals quite considerably. Injections of contrast medium are painful if the anaesthesia has been performed *lege artis*. On the other hand, the lower part of the back may be rather tender the day after the injection once the anaesthesia has worn off. The great majority of the patients preferred the examination to lumbar myelography.





|                   | Number<br>of<br>cases | Number of<br>changed<br>ligaments |     |
|-------------------|-----------------------|-----------------------------------|-----|
| Slight lordosis   | 80                    | 63                                | 79  |
| Moderate lordosis | 16                    | 84                                | 88  |
| Marked lordosis   | 53                    | 65                                | 123 |
| Total             | 149                   | 212                               | 193 |

Table 4. Incidence of changes in the three lower lumbar interspinous ligament in the presence of different degrees of lordosis.

All changes in the three lower interspinous ligaments roentgenologically diagnosed are included in the table and apart from cavities include rent and ruptures as well as flaccidity of the ligaments which latter may have another mechanical etiology than those mentioned earlier. Excluding flaccid ligaments the percentages show even more marked statistical difference viz. 80, 64 and 111 for slight, moderate and marked lordosis respectively. The results conform fully with the observation reported in the literature. It was thus possible to prove that *the incidence of pathologic interspinous ligaments in the three lower interspinous spaces is essentially higher in the presence of marked lordosis than in moderate or slight lordosis*.

#### HEIGHT OF THE SPINOUS PROCESS

In addition to increased lumbar lordosis BAASSTADT considered high (a large cranio-caudal dimension) spinous processes to be one of the primary causes of bone formation at the margins of the process. The same point was emphasized by STEHR, REINHARDT and JOSEPHANS. The present author also considered this point to merit closer examination in his series. The maximum height of all five lumbar spinous processes was measured in the lateral roentgenogram. Their combined height was then divided by the distance from the upper margin of the spinous process of L1 to the upper margin of the spinous process of S1. In backs with low spinous processes this index of the spinous height was low in the neighbourhood of 0.5 while in backs with high spinous processes it approached the value of 1.

The comparison would have been easier to perform for the combined height of the spinous process or their mean height but it was felt that this was not the correct procedure. It would have been necessary to employ the patient's body length as a factor of correction but longitudinal measurements

were not made. As a substitute for this procedure the author measured the spinous process distance  $L_3 \rightarrow L_4$ .

The table below shows the distribution of ligamentous changes in backs with different indices.

| Index       | Number of backs | Number of ligamentous changes | of changed ligament |
|-------------|-----------------|-------------------------------|---------------------|
| 0.00 - 0.25 |                 | 0                             | 11.8 %              |
| 0.26 - 0.60 | 11              | 8                             |                     |
| 0.61 - 0.65 | 23              | 6                             |                     |
| 0.66 - 0.70 | 31              | 26                            |                     |
| 0.71 - 0.75 | 36              | 28                            | 14.4 %              |
| 0.76 - 0.80 | 41              | 31                            |                     |
| 0.81 - 0.85 | 22              | 1                             |                     |
| 0.86 - 0.90 | 13              | 11                            |                     |

Table. The relationship between the mean height of the spinous process in the lumbar region and the changes in the interspinous ligament  $L_3 \rightarrow L_4$ .

The number of ligament changes in all the groups is close to the number of backs. Calculation of the percentage frequency of the changes for the low and the high half of the table respectively gives surprisingly uniform values. The final conclusion drawn can be no other than that the height of the spinous process does not appear to be correlated with the incidence of changes in the three lumbar interspinous ligaments.

#### THE CAPACITY FOR EXTENSION OF THE LUMBAR SPINE

All the earlier workers mentioned in the foregoing stressed the significance of narrowing of the interspinous space as the cause of compression lesions in the corresponding ligament. This is presumably how the ligamentous changes originated in the presence of increased lumbar lordosis.

The assessment of the degree of lordosis was doubtless subjective. Before beginning the contrast medium examination a lateral projection was made of the lumbar spine in maximal extension in all the cases in order to view this question from another and more exact standpoint. The minimum distance between the spinous process pairs  $L_3 \rightarrow L_4$  and  $L_4 \rightarrow L_5$  was measured and the millimeter values were grouped in the ranges 0—5, 6—10 and 11—15 mm. The following table gives a general idea of the total number of cysts, rents and ruptures in the interspinous spaces in question compared with the distance in maximal extension.

| Inter-spous<br>paces | Distance<br>mm | Number of<br>hanged<br>ligaments | Number of<br>interspous<br>paces |
|----------------------|----------------|----------------------------------|----------------------------------|
| L 3—4                | 0—5            | 0                                | 118                              |
|                      | 6—10           | 12                               | 88                               |
|                      | 11—15          | 2                                | 23                               |
| L 4—5                | 0—5            | 54                               | 137                              |
|                      | 6—10           | 2                                | 6                                |
|                      | 11—15          | 7                                | 30                               |
| L 5—S 1              | 0—5            | 29                               | 101                              |
|                      | 6—10           | 17                               | 71                               |
|                      | 11—15          | 10                               | 41                               |

*Table 6* The number of cavities, rents and ruptures in interpinous ligaments L 3—5 classified according to the distance between the pinous process in maximal extension

To make the comparison as lucid and reliable as possible the pinous process distance in maximal extension for all the three interpaces together was adopted as the point of departure for the following table

| Inter-spous<br>paces | Distance<br>mm | Number of<br>hanged<br>ligament | Number of<br>interspous<br>paces |
|----------------------|----------------|---------------------------------|----------------------------------|
| L 3—4 + L 4—5        | 0—             | 103                             | 356                              |
| + L 5—S 1            | 6—10           | 51                              | 23                               |
|                      | 11—15          | 19                              | 186                              |

*Table 7* The relation between the three different pinous process distance in the three lower lumbar interpace in maximal extension and the frequency of cavities, rents and ruptures in the corresponding ligament

The corresponding percentage when only the most severe changes—the ruptures—are taken into consideration for the smallest (0—5 mm) interpinous process distance was 13% for the 6—10 mm distance 8% and for the 11—15 mm distance 19%

The results of these calculations thus show the same trend as obtained for the posture of the lumbar spine and its effect on the changes in interpinous ligaments L 3—5. *The incidence of cavities, rents and especially ruptures was increased in the cases with a high degree of capacity for extension to which the 0—5 mm distance between the interspinous process corresponded.*

## DISPLACED SPINOUS PROCESSES

It was held by BUCKHARDT (1934) that whenever individual spinous processes appeared from examination of the lumbar spine to be displaced laterally there was reason to suspect a high degree of spondylosis deformans. BASTH wrote in 1910 that laterally displaced spinous processes reminded one of teeth that were badly set owing to insufficient space in the jaw. Such positioning of the processes would undoubtedly as a rule have been acquired in early childhood and may be considered as a physiologic variant which however disposed to pathologic conditions. The monograph by SCHMIDT & JUCHANNS (1937) mentioned that under developed and displaced spinous processes could be backache probably because of incorrect loading of the faultily working muscles. LEWIS (1937) found that the deviation of the spinous processes was of very frequent occurrence and proof of a strain at a certain level, evidence of altered dynamics of the spine and a sign of asymmetric muscular pull. The incidence of the level of deviation and pain in 198 cases was 78 per cent.

This deviation or lateral displacement of lumbar spinous processes is quite a common roentgenologic finding. In the present series of 229 cases 17 displaced spinous processes were demonstrated of 13, 10 of 11 and 23 of 13. A striking frequency of cavity and rupture like changes was disclosed already during the roentgenologic examination in these cases which suggested that a more detailed analysis of this observation was indicated. The table below gives a ready idea of the magnitude of this frequency compared with the corresponding incidence in the rest of the series.

| Interspous<br>ligament | Cavity                         |                                   | Rupture                        |                                   | Pathologic<br>ligament         |                                   |
|------------------------|--------------------------------|-----------------------------------|--------------------------------|-----------------------------------|--------------------------------|-----------------------------------|
|                        | Normal<br>spinous<br>processes | Displaced<br>spinous<br>processes | Normal<br>spinous<br>processes | Displaced<br>spinous<br>processes | Normal<br>spinous<br>processes | Displaced<br>spinous<br>processes |
| 1-3-4                  | 10                             | 6                                 | 18                             | 1                                 | 17                             | 41                                |
| 1-4-                   | 10                             | 1                                 | 33                             | 11                                | 33                             | 30                                |
| 1-5-1                  | 1                              | 1                                 | 33                             | 1                                 | 33                             | 33                                |

Table 8. The ratio between cavity and rupture in cases with displaced spinous processes and the rest of the series.

The table shows convincingly the great frequency and preponderance of cavities and ruptures in interspinous ligaments that are attached to displaced spinous processes compared with those in the sagittal plane. This is probably due to uneven loading within the ligament.

## LIGAMENT THICKNESS

It was established by RISSANEN that the structural principle of the interspinous ligaments was the same throughout the lumbar region but that there were nevertheless differences between the two lower ligaments on the one hand and the more cranially situated ligament on the other hand. One reason for this was that the supraspinous ligament ended in L 4 or L 3. This author noted further that the thickness of the interspinous ligaments of the L 1—2 space and often also the L 2—3 space usually did not change with age and that their thickness in a man of about 20 was about 1 to 2 mm and about 2 to 3 mm in the two lower spaces respectively. This was when the measurement was taken from the centre of the ligament usually the thinnest place. The thickness increased considerably on moving ventrally toward the ligamentum flavum and dorsally towards the supraspinous ligament or the aponeurosis of the back. A thickening of the ligament with age was in contrast almost a rule in the two lower ligaments and was most marked in the L 4—5 space in which thicknesses of 8 to 12 mm were fairly common. This thickening was considered by RISSANEN to be associated with the formation of cavities in the ligaments.

The present author's roentgenologic method made it possible to determine the thickness of the ligament simply by taking the transverse measurement in the roentgenograms. Interspinous ligament L 3—4 represented the cranial group. It is unfortunately not the most suitable ligament but it is the most cranial of those covered by the examination. Ligament L 4—5 represented the caudal group. The measurement was made at the level at which the ligament was thickest approximately halfway between the adjacent spinous processes.

The results obtained are given in the following table.

| Interspinous ligament | Thickness mm | 11—20 years | 21—30 years | 31—40 years | 41—50 years | 51—60 years | 61—70 years | Total |
|-----------------------|--------------|-------------|-------------|-------------|-------------|-------------|-------------|-------|
| L 3—4                 | 4—7          | 5           | 13          | 32          | 3           | 1           | 0           | 57    |
|                       | 8—11         | 4           | 15          | 34          | 1           | 8           |             | 84    |
|                       | ≥ 12         | 0           | 8           | 11          | 18          | 10          | 1           | 38    |
| L 4—5                 | 4—7          | 5           | 16          | 32          | 11          | 10          | 0           | 77    |
|                       | 8—11         | 4           | 17          | 34          | 9           | 6           | 1           | 111   |
|                       | ≥ 12         | 0           | 1           | 1           | 3           | 11          | 1           | 16    |

TABLE 9 Thickness of interspinous ligament L 3—4 and L 4—5 at different ages

A superficial examination of the table is sufficient to show that interpositional ligament I 3-4 is thinner on an average than the more caudal ligament. Moreover thick ligaments tend to be more common at the higher ages. Ligaments thinner than 4 mm were not encountered. The values reported by RISSANEN were on the whole lower than those for the present series. At least a partial explanation for the difference may be the routine histologic magnification which is about 10 per cent. The difference in tone between living and dead tissue may also be of significance in this connection.

These differences are easiest to see in the following figure which compares the percentual occurrence of the two extreme thicknesses for both interposures. The difference is not overwhelmingly great but it is apparent.

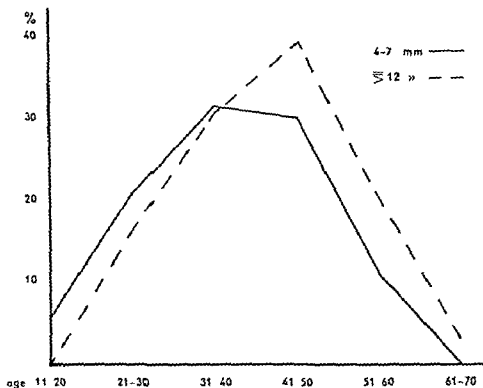


FIG. 3. The percentual distribution of 4-7 mm and  $\geq 12$  mm thick ligaments belonging to interposures I 3-4 and L 1-2.

The following table shows the distribution by ligament thickness of the total of avulsions and ruptures. Ruptures, as was also observed by RISSANEN, always display a minor or major cavity in the middle of the ligament.

| Ligament thickness | L 3—4               |                    | L 4—5               |                    |
|--------------------|---------------------|--------------------|---------------------|--------------------|
|                    | Number of ligaments | Number of cavities | Number of ligaments | Number of cavities |
| 4—7 mm             | 3                   | 3                  | 4                   | 3                  |
| 8—11               | 6                   | 10                 | 3                   | 3                  |
| ≥ 12               | 3                   | 3                  | 1                   | 1                  |

Table 10 The ratio between the incidence of cavities and the thickness of interspinous ligament L 3—4 and L 4—5

The two adjacent interspinous ligaments behave completely differently. In the interspace L 3—4 cavities are not numerous in the thinnest ligament and rare if the thickness is  $\geq 12$  mm. The cavity distribution in space L 4—5 again is remarkably uniform as regards ligament of different thickness. This observation that the ligament thickened considerably as shown by measurements taken on the outside when cavities originated is contrary to the present author's findings as regards the L 3—4 ligament nor does it altogether agree with the finding concerning the L 4—5 ligament. It is possible that the differences in the composition of the two materials have a role in this finding.

## VI THE DIFFERENT TYPES OF CHANGES IN THE THREE LOWEST INTERSPINOUS LIGAMENTS THEIR INCIDENCE AND ROENTGENOLOGIC DIAGNOSIS

Mention has already been made in passing of the type of ligamentous changes that may be revealed by roentgenologic contrast medium examination. The roentgen findings in normal ligaments were also described in brief. In the following each group is discussed separately with special reference to the roentgen diagnosis in the examination of the ligaments.

### NORMAL LIGAMENTS

Normal interspinous ligaments were encountered in the three lower lumbar inter spaces in 21 control cases (43 per cent) and in 69 of the cases with lumbar pain (36 per cent). The difference was distinct, but the materials are not fully comparable as the mean age of the controls was lower than of the group with back pain. This point will be considered again in discussion of the correlation between back pain and interspinous ligament change. *After a paraligamentous contrast medium injection normal interspinous ligaments appear in the anteroposterior view as medially sharply defined but sometimes slightly irregular, equally broad or somewhat spindle-shaped contrast medium defects which are cranially and caudally delimited by two adjacent spinous processes.*

With thin spinous processes the upper pointed long and narrow margin of the process often appears to dip into the ligament and the contrast medium consequently does not reach halfway up the height of the lateral boundary (Fig. 4). The lower margin of the spinous process is often considerably broader and arched and the contrast medium reaches the lateral contour of the process low down and caudally. The same is true cranially if the transverse section of the process is piriform and the maximum transverse measurement in the frontal plane is consequently large (Fig. 5). The interspinous ligaments are probably attached to their spinous process at level which appear to vary with the different forms of the fixed surface of attachment; it is possible that mechanical tenacity conditions have some





Fig. 4

Fig. 5

Fig. 4 Normal findings. Spinous processes are thin and thin. Extremely breaky contrast medium structure. Upper point of spinous process more on L4 lig into the ligament and the contrast medium reach only halfway up the contour of the spinous process.

Contrast medium has no contact with spinous process L5 which appears to be embedded in a tissue impenetrable to the medium and probably of fibrous origin.

Fig. 5 Normal findings. Spinous processes L3 and 4 are rounded and broad. Graciously sufficient attachment surface for the interspinous ligament at the upper margin of the spinous process. Contrast medium therefore reach the spinous contour much better than in Fig. 4.

influence. In Fig. 4 the contrast medium has not established any contact at all with the contour of spinous process of L5, the process as a whole is evidently embedded in tissues impenetrable to the contrast medium and it is possible that the interspinous ligament encircles it completely.

RISSEN pointed out that the ligament was thinnest in its central part. According to him the interspinous ligament viewed in the projection of the roentgenogram was shaped somewhat like an hour glass.

The roentgenologic form however is usually the exact opposite. This may be due to technical factors of projection but the similar appearance of the ligaments at different depths does not support this suggestion. The

cause of the difference in ligament form is possibly contingent upon the difference in tissue turgor.

The medial sharp contour originates when the ligament prevents the contrast medium deposit from protruding right up to the median line. Laterally on the other hand there is no impassable obstacle and consequently the contour of the contrast medium is considerably irregular in this direction. The more or less streaky structure seen laterally and caudally in particular appears to be dependent on the penetration of the contrast medium between the dorsal muscle fibres (Fig. 4). Another example of a normal roentgenologic finding may be seen in Fig. 6.

#### TRAUMENTS IN DISTALCED SPINOUS PROCESES

Spinous processes with a laterally divergent course were encountered in the present material as follows: 17 of L 3, 10 of L 4 and 23 of L 5. In one case all 3 spinous processes were displaced and in 7 cases 2 were deviated.

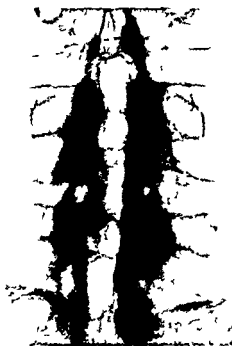


Fig. 6. Normal finding. Inter spinous ligament 14—rather thin and of uniform thickness. 15—S1 ligament—somewhat asymmetric.

The commonest type of deviation was that the caudal part of the spinous process was displaced laterally while the cranial margin was usually on the median line and level with the main row of spinous processes (Fig 7)

Such displacement of the spinous process naturally also affects the attachment of the ligament to the spinous process which becomes asymmetrical and displaced laterally. The half of the ligament that is situated on the side to which the spinous process is displaced is attached to the lower margin of the process while the attachment of the opposite half extends higher up (Fig 7). The upper margin of the spinous process may sometimes deviate from the median line (Fig 8) with the vertebra as a whole simultaneously rotated slightly to the left. The ligament attachment is asymmetric in this case too.



Fig. 7



Fig. 8

Fig. 7 Left half of inter-spinous ligament L4-L5 attached cranially to the lower margin of the displaced spinous process L4 while the right half is attached to the lower part of the spinous process on the side.

Fig. 8 Displacement of spinous process L5 with right sided deviation of its upper margin. Contralateral lamina adheres on the right side to the deviating spinous process margin on the left side it achieves contact with the process considerably lower down.

## CAVITY FORMATION

According to BAASTRUP (1933) as early as 1824 MAYER demonstrated the presence of true joints with an articular cavity and a synovial membrane between the spinous processes of the lumbar spine so called Diarthroses inter spin. Mayer LICK (1904) describing the normal anatomy of the inter spinous ligament stated that a uni or multilocular joint cavity had sometimes been seen in them. He did not mention however whether he regarded it as a pathologic or normal phenomenon. BAASTRUP'S conclusions concerning inter spinous ligaments were for the most part arrived at indirectly through an analysis of roentgenologic changes pertaining to the spinous process. But he offered no personal observations throwing new light on the circumstances connected with these cavities.

It was pointed out by BRANFORD (op cit) that bursae and arthritic changes develop readily between the spinous processes in the presence of increased lumbar lordosis. Adventitious bursae were encountered especially in the tall slender visceroplathic type of skeleton with a long narrow flexible spine. WINDHOLZ (1937) studied the spinous processes roentgenologically and made anatomic comparisons. He stressed specifically the common occurrence of exostoses on the lower external margin of the spinous process bony projections that were of varying size and shape. Between the exostose and the adjacent spinous processes he encountered true joints with cartilage covered articular facets and a fibrous articular capsule. They were found regularly in cases in which the static mechanical functions of the spine had sustained change caused by pathologic processes.

The first description of a case of lumbar inter spinous cavity studied histologically that the author has been able to find in the literature is the one published by FALLON, LEGER & AHRAS (1949). They stated that the spinous process is a poor relation of the rich vertebral pathology. They found in a case in which spinous processes L3—4 had been resected because of back pain and new bone formation at the margins of the spines an articular cavity with cartilage covered surfaces and capsule in the interspace L3—4. Histologic examination of the preparation revealed that there was a cavity unlined with cell elements in the middle of a block of fibrocartilage.

YAMADA, NISHIWAKI & YASUKAWA (1954) performed spinal fusion on a 29 year-old heavy labourer with intractable low back pain which they attributed to morbus Baastrop. The man had previously been exposed to hyperlordosis trauma. A sketch showed the presence of cavities between spinous processes L3—4 and L4—5. They called these cavities bursae.

The morphology of inter spinous ligaments in human subject of different ages was studied histologically by SCAPINELLI (1935). He demonstrated in an interspinous ligament of a kyphoscoliotic patient of 39 a cavity with a wall reminiscent of that in a mucosal bursa or an articular synovial membrane.

The investigation performed by RISSANEN showed that notable changes occurred with age in the interspinous ligaments some of them visible to the naked eye. One of these changes was the formation of cavities. The older the person the greater was the likelihood of a cavity in the middle of his interspinous ligaments. This cavity formation was more common in the L 4—5 space than in the L 5—S 1 and L 3—4 spaces. Incipient cavity formation was often seen in the other three spaces in younger persons and in older subjects in the upper spaces as well. If there were cavities in several space they were usually largest in the L 4—5 space.

Cavities were found to begin to originate immediately after the 20th year of age. Not a single case was found in subjects under 20 in RISSANEN's series. It seemed as if the centre of the ligament first became soft and slack and the tendinous tissue disappeared from the area later to be replaced by an empty cavity or one partly filled with fat and loose connective tissue appeared at its site. In younger subjects and in those in which the cavities were incipient the walls generally were irregular and rough. In older subject and where the cavities were larger the wall usually grew smooth. Externally the ligament may appear intact in spite of it containing a cavity.

In the series mentioned above a true kinking spine was not an absolute precondition for the formation of cavities. Morbus Baastrup cases are relatively rare and were only established a few times with certainty in his material. Cavity formation was most marked in the lower lumbar space where the lordosis also was most evident. These observations lend support to the assumption that rubbing of the spinous processes against one another either directly by destroying the tissue or indirectly by causing degeneration had a role in the origination of cavities. It was very common in subjects over 50 for the cavities in the lowest interspinous ligaments to be bounded on the sides of the spinous processes by tissue resembling hyaline cartilage cell colonies. The cavities originating inside the interspinous ligament together with the surfaces of adjacent spinous processes thus made at least in later years of life joint like formations with synovial membranes and articular capsule. There must consequently have been notable chafing and pressure between the bony surfaces of the spinous processes as has been proved in experimental studies of arthrogenesis (KROMPECHER & GOLTTNER 1938, KETTUNEN 1939).

The present author was able to demonstrate cavitation roentgenologically in 14 cases and 51 ligaments, i.e. in 19 per cent of the total cases. The cavities were distributed fairly evenly between the three lower spinous interspaces but with a slight preponderance for the middle of these three. Cavities were very common in cases with displaced spinous processes, 19 of the 51 cavities were established in this group although such spinal processes only constituted slightly over 10 per cent of the total. Cavities in the cases with displaced spinous processes occurred primarily in the age group 21—30 and 31—40 years; the slightly higher ages were better represented in the other cases.

The difference between the incidence (75 per cent) of cavities in the age group 31—40 years in RISSANEN's material and the figure of 19 per cent for the present series is surprisingly large. It may naturally be due in part to dissimilarities in the two materials. Another factor may be that the frequency of ruptures in the present series is essentially greater than that in the autopsy material. It is likely that the present author diagnoses ruptures many ligamentous changes that RISSANEN would have classified



Fig. 9

Fig. 9 Cavity in interspinous ligament L 4—5 which is shown roentgenologically by an irregularly delimited round spot of contrast medium in this interspace.



Fig. 10

Fig. 10 Cavitation spinous process L 4 levitated caudally to the right. A long, heavily lying along the left ligament contour fills a large part of the interspinous ligament L 4—5.



Fig. 11a



Fig. 11b



Fig. 11c

Fig. 11 a) Cavity in 2 mm in diameter represented by a round spot of contrast medium located in interpinous ligament L3-4 and a spot of contrast in diam  $4 \times 9$  mm in ligament L5-S1. b) Tomogram 3 cm cut Cranial cavity loc in this plane the caudal cavity is more rounded in shape than in (a) owing to moderate extension of the lumbar spine. c) Tomogram 4 cm cut Cranial cavity begins to appear.

as cavities. It is perhaps not always certain to distinguish between them by roentgenologic means nor is it highly significant since the difference between them may be subtle. A third reason for the difference may be that the cavities were contained between bilateral fairly intact ligamentous parts which prevented the contrast medium from penetrating. Fourthly, a cavity filled completely with fatty tissue may sometimes have failed to produce roentgenologic evidence of its nature. All these possibilities are conceivable and in full conformity with observations on cadavers.

The diagnosis of a cavity by contrast examination of lumbar interspinous ligaments is based on the demonstration of a rounded (Fig 9) or somewhat elongated (Fig 10) spot of contrast medium within the contrast free one produced by the ligament. The shape is naturally dependent also on the degree of flexion or extension of the lumbar spine. This may be seen also in Fig. 11 in which the tomograms were taken on another table than the one employed for the ordinary contrast roentgenogram resulting in a certain degree of extension of the spine. The accumulation of contrast medium may be only 1 to 3 mm in diameter (Fig. 11 the L 3-4 interpace and Fig. 12) but also as much as 6 to 10 mm (Fig. 13). In tomograms with 1 cm between the



Fig. 1. (C. C. C.) A rounded relatively small cavity in interspinous ligament L 4-5





Fig. 13a



Fig. 13b

Fig. 13a) Cavitation. A rounded, medium-sized cavity in ligament L3-4; a large, sharply delineated longitudinal cavity in interface L4-5. Very thin layer of sacral ligament.  
b) Tomogram. 3 cm cut. A streak of contrast medium caudally to the right in ligament L4-5 correct. The spot of medium with the ligament lying out of the beam at relatively a right in the right half of the ligament.

sections the cavity is sharply delineated in 1 or at the most 2 cuts showing that its boundaries in an anteroposterior should be roughly the same as in a lateral direction. The contour may be completely regular (Fig. 13) but is more often irregular (Fig. 9) obviously depending on the quality of the surface within the cavity.

The spot of contrast medium usually adheres more or less intimately to the general contrast deposit on one side. This shows that the ligament is thin and that the contrast medium has probably penetrated the ligament from that side. A frequent indication of where this has occurred is a streak of contrast medium connecting the contrast medium accumulations outside and inside the ligaments. Such finer details can best be analysed with the aid of tomography (Fig. 13). Figs. 10 and 17 are examples of the frequent

occurrence of cavities in displaced spinous processes. It is often quite difficult to classify the penetration of contrast medium in a ligament. Borderline cases occur primarily between cavities and partial ruptures. This problem will be discussed in more detail in connection with the latter type of change.

#### RUPTURES IN THE LIGAMENTS

NEWMAN (1952) pointed out that injuries resulting in the springing apart posteriorly of two vertebrae frequently occurred in everyday life. This may happen in a fall on a slippery surface with the legs stretched out in front, a fall downstairs or from a horse or bicycle, or a skating accident. A similar strain to the spine may occur when a heavy weight is lifted with the spine flexed but the knees straight. The result of violence of this nature, when of sufficient force, is to tear among other the supraspinous and interspinous ligaments. The region of the spine affected is the one where the mobile lumbar spine meets the immobile pelvic girdle, and this is generally the lumbosacral junction. NEWMAN called the result of a trauma of this kind *pruned back*.

Torn or inefficient interspinous ligaments in the lower part of the lumbar spine were often encountered by KALLIO (1960) in disk operations. Since the autumn of 1957 he has carried out cutaneous ligamentous reconstruction in some of these cases using skin from the edge of the incision as repair material. A similar method has been tested experimentally in rabbits by JONSSON (1958).

RISANEN established that the interspinous ligaments of children are intact. This was not always the case with adults who relatively often have defects in certain parts of the ligament system. He considered that the ligaments must therefore be ruptured in one way or another and called these defects *ruptures*.

The ruptures found in interspinous ligaments were of two types.

1) Partial ruptures which showed on either the right or the left side of the ligament as a rent across one half of the ligament. The other half of the ligament was intact although generally much slacker than usual. The rent extended to the cavity that was almost always present in the centre of the ligament in cases of this type.

2) Complete ruptures with a fairly wide rent through the ligament, the opening was either empty or filled with fatty tissue.

The rupture was typically and always localized at a certain site, a constant one in the interspinous ligament. This point of predilection was

the medial portion of the ligament the ventral and dorsal part being usually intact. The complete ruptures were usually oval in form with an anteroposterior axis. In some cases they were rounded. They were well demarcated and sharply defined from the rest of the ligament.

It was reported by NEWMAN that the supra- and interspinous ligaments ruptured completely in the space between the spinous processes when subjected to trauma although apart from ketcher he advanced no proof of this. RISSANEN on the other hand found no signs of rupture of the supra- and interspinous ligaments in any of his 306 cases even on microcopy. In the rupture of the interspinous ligament that he noted the posterior part was always intact and only the medial part was torn in the area where the fibre bundle passes obliquely between two bony surfaces of different vertebrae.

Histologic examination showed that the rupture of the interspinous ligament always occurred in tissues showing varying degrees of degeneration but never in healthy tissue. The more interesting of the degenerative changes was cystic degeneration. It had morphologic features similar to those seen in ruptures of the tendon of the supraspinatus in the shoulder reported by SCHAEFER (1936). The changes were also in some respects similar to those occurring in ruptures of different tendons reported (BJORKROTH 1943, DAVIDSON 1946, ORELL 1958).

Ruptures were found by RISSANEN in about 21 per cent of subjects over 30 years of age, two thirds of these were partial and one third complete. The first case, an isolated one, was found in a subject of under 30, there was a peak in the 30—40 year age group, above which the incidence was relatively even. The present series showed a considerably higher rupture frequency, i.e. 47 per cent, of which 21 per cent were partial and 26 per cent complete. The considerable difference in the incidence will be discussed later.

Twenty-two of the cases of rupture were women and 87 men. Since in the total material there were 63 women and 166 men, the relative incidence among women was clearly lower than among men, 33 per cent against 52 per cent respectively. The ratios were reversed in the autopsy material but the difference was no more than 7 per cent.

a) *Partial ruptures* Partial ruptures were demonstrated roentgenologically on the right side in 28 cases and 31 ligaments and on the left side in 21 cases and as many ligaments, i.e. in all in 49 cases or 21 per cent. The incidence was highest in the L4—5 interspace which displayed more ruptured ligaments than all the other interspaces together. The pathologic changes were more numerous in the lumbosacral interspace than in the L3—4 in

terspace. The percentual distribution of the cases in age groups of a reliable magnitude from the statistical sample is fairly uniform and is around 20 according to the table below

| Years | Total number of cases | Number of cases with partial rupture |    |
|-------|-----------------------|--------------------------------------|----|
| 11—20 | 9                     | 4                                    | 44 |
| 21—30 | 12                    | 7                                    | 17 |
| 31—40 | 77                    | 17                                   | 22 |
| 41—50 | 71                    | 14                                   | 20 |
| 51—60 | 27                    | 6                                    | 2  |
| 61—70 | 3                     | 1                                    | 33 |

Table 11 Number of cases in the different age group and the incidence of partial rupture

*Partial ruptures appear after the injection of contrast medium as streaks which run parallel but not quite rectilinear to the superior or inferior surface of the vertebral bodies (Fig 14). The streaks depart from the main contrast medium deposit at the outer contour of the ligament and penetrate to the median line of the ligament where they often increase in breadth (Figs 15)*



Fig. 14

Fig. 14 Partial rupture. Streak of contrast medium on the right side of intervertebral ligament L5—S1 suggesting rents in its structure



Fig. 15

Fig. 15 Partial rupture. Lumbo-sacral ligament has an irregular right-sided contour. A streak of contrast medium increases in breadth toward the centre of the ligament representing a partial rupture



Fig. 16

Fig. 16 Partial rupture. A typical finding to the left in the L4—5 intervertebral disc at L4—5.



Fig. 17

Fig. 17 Partial rupture. A long oval spot of contrast medium adherent to the right canal wall in the L4—5 intervertebral disc, indicative of a hollow partial rupture. Large cavity in the L5—S1 ligament to the left.



Fig. 18 Complete rupture. Contrast medium accumulation in the canal, connected by an oblique bridge of medium, which penetrates into the ligamentous space at L4—5 and represents a rupture.



Fig. 19a



Fig. 19b

Fig. 19a. Complete rupture. Transverse bridge of contrast medium in ligament 13-4 and 14-5 revealing rupture in these ligament. 1) Tomogram 3 cm out. Change are more distinct than in (a)

and 16. The streaks may also be of equal width but are usually of different length and one to three in number. The ligament contour is almost always slightly irregular on the side of the departure of the streaks and sometime only an irregularity in which the contrast density is increased represents the partial rupture (Figs. 17 and 21).

It is often difficult to draw a line between partial ruptures and cavities. In Figs. 15 and 16 the streak of contrast medium is distinctly broader toward the centre of the ligament which suggests the presence of a cavity. On the other hand it has to be admitted that the contrast medium spot which represents the cavity sometime displays a canal communicating with the surface of the ligament (Fig. 13). All of RISSANEN's rupture cases, both partial and complete, had a minor or major cavity in the middle of the ligament. The obvious explanation is simply that the boundary between the two types of change is ill defined. When the contrast medium spot is large and there is no communication with the exterior or when it is quite small it is appropriate to speak of a cavity. When the contrast medium spot is



Fig. 00a



Fig. 01



Fig. 0c

Fig. 0a) Complete rupture. A large rupture in the lumbo-sacral ligament. It fills the lumbar contrast medium deposit. b) Tomogram 3 cm cut. Centrally located dense accumulation of contrast medium in the ligament outlines a cavity. c) Tomogram 4 cm cut. Rupture of different height owing to the depth of the section.

small and the communicating canal is about as wide as or wider than its diameter. The principal pathologic feature is a rupture. Certain of the variants of the findings tend to cause difficulties in interpretation.

b) *Complete rupture*. This type of rupture was diagnosed in 60 cases and



Fig. 1. Complete rupture. A rupture of irregular shape in interpinous ligament L. 1-2.



Fig. 2a



Fig. 2b

Fig. 2. a) Complete and partial rupture. A complete rupture in the lumbo-sacral interpace and partial rupture on the left side in interpinous ligament L. 4-5.

b) Lumbosacral interpace. Rupture more distinct than in (a).



Ligaments i.e. 26 per cent. The majority of the rupture in this group were also in interspace L4—5 followed closely by the lumbo sacral interspace while interspace L3—4 contained by far the smallest number of rupture. The incidence shows a certain increase with advancing age as is evident from the following table

| Years | Total number of cases | Number of cases with complete ruptures |    |
|-------|-----------------------|--|----|
| 11—20 | 1                     | 1                                      | 11 |
| 21—30 | 4                     | 10                                     | 4  |
| 31—40 | 77                    | 15                                     | 20 |
| 41—50 | 71                    | 3                                      | 3  |
| 51—60 | 27                    | 11                                     | 41 |
| 61—70 | 3                     | 0                                      | 0  |

Table 1\* The number of cases in the different age groups and the incidence of complete ruptures

Complete ruptures are fairly easy to diagnose after the injection of bilateral paraligamentous positive contrast medium. This forms a bridge between the two contrast medium deposits which originate when the contrast penetrates the ligament as the site of the rupture fills. The direction may be oblique (Fig. 18) or transverse (Fig. 19).

The breadth usually exceeds half the distance between the adjacent spinous processes and the contrast medium may even fill in the interspace completely (Fig. 20). The structure of the contrast medium may be homogeneous or lightly streaky or blotched and the bridge is sometime considerably irregular in shape (Fig. 21). Complete and partial ruptures sometimes occur in the same subject (Fig. 22). The cavity that ought to be demonstrable in the centre of the ligament can be discerned clearly e.g. in Fig. 20.

The incidence of cavitation in the present series was much lower than in RIAXENS material which was exactly the reverse for ruptures as has been mentioned earlier. A possible tendency to diagnose ruptures too frequently may very well be due to the fact that large cavities are surrounded by a very thin and degenerated ligamentous wall that it may be permeable to contrast medium and also easily damaged during operative procedures.

methods. The exactitude is probably in most cases not the same as RISSANEN achieved, but his dissecting method required cadavers for the assessment of ligament flaccidity; however, both the *in vivo* methods should be superior to the preparation of cadavers.



Fig. 25a



Fig. 25b

Fig. 25 a) Relaxed ligaments. Changes typical of a flaccid ligament in the L 3-4 interpace. Partial rupture to the right in ligament L 4-5. In lumbo-sacral interpace a large cavity with irregular margins, suggesting concomitant ligament flaccidity. b) Tomogram 4 cm. caud. L 5-S 1 cavity in a very diffuse ligament contour well known.

# VII DISK DEGENERATION TRAUMA OF THE LUMBAR SPINE AND CHANGES IN THE INTERSPINOUS LIGAMENTS

## DISK DEGENERATION

BAASTROP (1953) was interested in the connection betweenpondylosis deformans and changes at the margins of the pinus proci e a cause of pain He pointed out at the same time that it is possible to encounter *osteoarthritis of the spinous processes* without associated pathological changes in the vertebral bodies

The author considered in analysis of the correlation of disk degeneration and changes in the inter spinous ligament to be of interest for the purpose the conventional lumbar roentgenogram in the material as a whole were examined for signs of the former narrowed inter spinous spaces marginal deposits and sclerotic edge of vertebral bodies as well as displacements between vertebrae were taken as sign of instability The cases were then distributed into four groups according to the findings normal = 0 and I II and III degree of degeneration The table below shows the number of cases in the four groups and the distribution of cases with ligamentous changes

| Degree of disk degeneration | Total number of cases | Cases with ligament changes |    |
|-----------------------------|-----------------------|-----------------------------|----|
| 0                           | 78                    | 11                          | 6  |
| I                           | 87                    | 6                           | 11 |
| II                          | 3                     | 3                           | 6  |
| III                         | 1                     | 6                           | 0  |

Table 17 The incidence of changes in inter spinous ligament in the total material Cases distributed according to the degree of disk degeneration in the lumbar spine

It appears from the table that the changes in the three lower inter spinous ligaments do not seem to have any connection with disk degeneration in the lumbar spine or in general with its severity It has been shown as previously

stated that ligamentous changes are of a degenerative character partly the result of heavy local mechanical strain caused by anatomic factors as well as by certain other more general factors affecting the organism as a whole. Disk degeneration is as the name implies also caused by degenerative changes but originates independently of them in the interspinous ligaments. *The changes which are similar in character in the various sections of the spinal column thus obviously have a different etiologic background.*

The connection between prolapse of disk and changes in the interspinous ligaments have been the subject of lively discussion in the literature. NEWMAN stated that a flexion injury to the posterior structures of the vertebral column if carried beyond the point at which the neural arches of two vertebrae are torn asunder will separate the vertebral bodies and damage the posterior longitudinal ligament and the annulus fibrosus which bind the bodies together. It is rational to regard disk prolapse as secondary to ligamentous damage. In support of this assertion he referred e.g. to FRUHING & HIRSCH (1949) who showed by examination of 100 disks at necropsy that in the lower lumbar spine it is almost invariably the posterior arc of the disk that is damaged. This would seem to suggest that the injuring force separated the vertebrae posteriorly. If injury to the annulus were due to compression torsion or grinding the damage would not be confined to the posterior quadrant. Further proof might be found in NEWMAN'S experience in operation for prolapsed disk: a torn or inefficient suprapinous ligament and an unstable vertebra were almost constant findings.

A similar reason for disk prolapse was advanced by HACKETT (1957): «When there is relaxation of the supra- and interspinous ligaments an increased pressure on the intervertebral disks is permitted. This pressure may force the nucleus pulposus through the posterior longitudinal ligament giving rise to the increased symptoms of radicular pain».

Of all the ruptures in the lumbar interspinous ligaments in RISMANLYN'S material 92 per cent were situated in the two lowest ligaments. The majority of all disk prolapses were also situated in the two lowest vertebral interspaces. He therefore suggested that there may be some causal connection between these two facts. The prolapse usually occurs in the dorsal part of the disk and hence from the purely mechanical point of view yielding of the most posterior parts of the spine should be of considerable even decisive significance as a precondition for the prolapse.

A connection between disk prolapse and changes in the interspinous ligaments was also suggested by HYD (1959) but his reasoning was in direct opposition to that of the three authors mentioned earlier. He considered that

chronic overstretching of the interspinous ligament occurs in connection with degeneration of the disk in consequence of the narrowing of the corresponding intervertebral spaces. Provided that the small intervertebral joints are intact the two corpora try to approach one another and the two corresponding spinous processes concurrently draw apart. This displacement takes place around a frontal axis the site of which is governed by the location of the articular surfaces on the articular process. The result is stretching of the supraspinous and interspinous ligaments and it is conceivable that such distension or trauma may produce degeneration of the ligament.

Were a pathologic or above all a ruptured ligament the cause of disk prolapse or vice versa ligaments of this type ought to be a very common finding in homologous interspinous spaces. Those cases of the present series that had been subjected to a disk operation ought to lend themselves excellently in illustration of this question.

These cases total 83. Prolapse of the disk was established in the L 4-5 interspace in 2 cases and in one of these in the L 4-5 interspace as well in a further 43 cases; this interspace was the site of the prolapse which was eliminated. The lumbo-sacral disk was the starting point for a prolapse in 38 cases. The following table gives a general idea of the occurrence of pathologic ligaments in disk prolapses. The prolapses in interspaces L 3-4 and L 4-5 in the same case are entered in the table as occurring in separate cases.

Analysis of the figures shows that prolapse in the lumbo-sacral interspace are accompanied by a significantly greater number of changes in the interspinous ligaments than those in the immediately cranial interspace. This however pertains solely to the heterologous change; homologous change

| Interspinous<br>ligament | Prolapses       | Changes in<br>homologous<br>ligament |                 | Changes<br>heterologous<br>and homologous<br>ligament |                 | Changes<br>heterologous<br>ligament<br>only |                 | Total<br>ligament<br>changes |                 |
|--------------------------|-----------------|--------------------------------------|-----------------|---|-----------------|---|-----------------|------------------------------|-----------------|
|                          | No. of<br>cases | No. of<br>cases                      | No. of<br>cases | No. of<br>cases                                       | No. of<br>cases | No. of<br>cases                             | No. of<br>cases | No. of<br>cases              | No. of<br>cases |
| L 3-4                    | 2               | 1                                    | 50              | 0   | 0               | 0   | 0               | 1                            | 50              |
| L 4-5                    | 46              | 8                                    | 17              | 1   | 20              | 3   | 20              | 26                           | 51              |
| L 5-S 1                  | 38              | 6                                    | 16              | 10  | 26              | 16  | 42              | 32                           | 84              |
| Total                    | 86              | 15                                   | 17              | 11  | 46              | 19  | 62              | 59                           | 161             |

Table 14 Incidence of change in the three lower interspinous ligament in homologous and heterologous interspinous space in 86 cases of disk prolapse operatively treated

were established in practically the same degree in both inter spaces and in about 40 per cent of the prolapsed cases. Flaccid ligaments and ruptures in the lumbo sacral interspace are over represented in this part of the series. A comparison between the incidence of ligamentous change in the prolapse material and in the rest of the cases with back pain gave the following ratio  $79/86 = 69$  per cent  $73/104 = 70$  per cent respectively.

Disk prolapse operatively verified and a pathologic interspinous ligament at the same level thus displayed a congruence in 40 per cent of the cases. But in addition over half the cases with homologous ligamentous change also presented concomitant heterologous ligament changes. Heterologous ligament changes alone occurred in 30 per cent of the prolapses. Taking this into consideration heterologous ligament changes were encountered in about 40 per cent of all cases of prolapse. The remaining 30 per cent of the prolapses were associated with intact interspinous ligaments.

The congruence between disk prolapse and changes in the corresponding interspinous ligament often established operatively observed by NEWMAN and KALLIO was thus no greater than 40 per cent in the present series. There was concurrently an incongruence in regard to the ligamentous changes which rose to 50 per cent and furthermore every third case had normal interspinous ligament. The authors' studies consequently do not support NEWMAN'S and HACKETT'S arguments concerning ligamentous changes as the cause of disk prolapse. It would appear very probable that their assumptions are incorrect with the same applying to HYND'S argumentation.

The incidence of pathologic ligaments was 69 and 70 per cent respectively in the cases with disk prolapse operatively verified and the unoperated cases with back pain. This comparison is in full agreement with the investigation results discussed on pp 49—50 and also speaks against an etiologic connection between disk prolapse and altered interspinous ligaments.

#### TRAUMA

Minor signs of trauma of the lumbar spine as the reason for morbus Baastrup have been reported in the literature. They were mentioned earlier by SCHUMANN & TRAUTMANN who in post traumatic cases obtained low back pain in the lumbal region which appeared spontaneously when the back was extended. These cases had marked bony growths on the spinous processes and narrowed inter spaces between the processes. The reason for the clinical sign these authors stated was the static changes in the lumbar spine after traumata caused by faulty loading.

All the author's patients were questioned closely about any earlier trauma of the lumbar spine. No such injury was recalled by 115, while 81 was recalled in the affirmative. Only definite cases were included in this group. Some two-thirds of the 81 cases reported that they had lifted too heavy a load, about one-third that they had performed a movement that was unnatural and mostly that they had slipped and fallen or that a leg had given way under a load on the back. Only isolated cases had been exposed to direct trauma to the lumbar region. All 81 cases referred their back pain to the same mechanism mentioned.

The injuries were all of such types that they could well have caused a lesion of the interspinous ligament. There is reason in this context to recapitulate the electromyographic studies reported by FLOYD & SILVER (1951). The authors stated that in the standing position there is no contraction of the erector spinae muscles, but as the spine begins to bend forward the muscles contract until full flexion is reached. At this critical point the muscles again relax leaving the spine supported by the ligament. It is at this stage when the spine is in a position of flexion with the erector spinae relaxed that the ligaments of a damaged segment either fail to take the strain or in doing so produce pain.

Ninety-five changed interspinous ligaments were established in the 81 cases with a history of trauma, i.e. 117 per cent. The 148 cases without trauma covered a total of 117 changed ligaments, i.e. 79 per cent. This significant percentual difference seems to suggest that *lumbar spine trauma of the type mentioned tend to produce ligamentous changes radiologically verifiable in the three lower interspinous ligaments*.

## VIII CHANGES IN THE THREE LOWER LUMBAR INTERSPINOUS LIGAMENTS AND LOW BACK PAIN

Compression of nerve roots in consequence of prolapse of the intervertebral disk is the commonest cause of sciatica. It is not many decades since this was stated but it is of great significance in the interpretation of low backache is contested by none. It appears, however, that this has resulted in such great interest in the pathology and clinical features of intervertebral disks that the possible influence of the region behind the spinal canal on conditions in the lumbar spine may have been overlooked completely.

For instance, a great number of ligaments in the most posterior part of the lumbar spine may be the possible site of symptoms. This is apparent e.g. from the fact that LERICHE (1930) was able to demonstrate a profuse supply of nerve endings in articular ligaments and from COMROE'S observation that the ligament is after the periosteum the human tissue that is most sensitive to pain.

However, the theory of the ligamentous origin of pain is not completely without advocates. As early as 1916 MACNULTON (1944) stated before the American Roentgenological Society that low back pain and sciatic pain were to be attributed more to the strain of ligaments of the lower back than to any other cause. He mentioned in his paper that inflammatory and degenerative change occurred equally often in the ligaments and joints of the lumbar spine as in a knee which in such cases gave clinical signs of a different type. The weight of the body loads the joints and ligaments behind the spinal canal. On bending forward some of the load on the vertebra and disks is relieved but the posterior ligaments are stretched. If pathologic degenerated ligaments lacking elasticity are involved such movement may produce pain either from overstretching or from minute tears produced in the fibrous structure.

Whenever it is possible to demonstrate pathologic changes in the spinous contours there is reason to suspect that they or perhaps rather the change in the interjacent or adjacent soft parts are the cause of the lumbago which it is sooner or later (BAASTRUP 1933 b). In 1940 this author stressed further more that the most frequent direct causes of pain are undoubtedly the



matomata reactive edema and perhaps sometimes actual irritative processes in the periosteum of the ligaments as well protracted muscle contraction was then the most common secondary cause. He regretted that none of these factors could be demonstrated roentgenologically.

FRANCK (1943) in 2145 patients with back pain found 14 with a lesion of the spine as the cause of their symptoms all of them having a marked tenderness on pressure between the affected spinous processes. Eleven of the eleven operated on these patients had suffered repeated attacks of lumbago especially on bending forward and were resistant to every kind of expectant treatment. According to FAULON, LECER & AHRAS pressure on the spinous processes or especially on the inter spinous spaces cause a more or less strong however not constant pain in inter spinal neurothrosis their name for kissing spine. The roentgenologic examination offers the best diagnostic aid.

NEWMAN worked at a clinic dealing with backache where cases with infective or neoplastic signs had been eliminated. He considered that 20 per cent of the cases with pain in the lumbosacral area had symptoms deriving from rupture of the posterior ligaments of the spine including sometimes the posterior longitudinal ligaments and annulus fibrosus. He established in these cases pain in the mid line on palpation either at the space between the spinous processes of the fifth lumbar and first sacral vertebrae or between the fourth and fifth lumbar vertebrae. JOSEPHAN was of the opinion that deep non radiating median low back pain which he established in 60 of 80 lumbago cases were caused by M. Brastrup. Pinching and tearing in the soft parts between the spinous processes were in his opinion the cause of the symptoms.

The expression ligament relaxation was used by HACKETT (1937) as an idiom for pathologically altered ligaments without any detailed verification of the more exact nature of these changes. He was of the opinion that this relaxation was the reason for the back pain in the majority of all his patients and a previously mentioned also of importance in the genesis of disk prolapse.

This review of the literature shows that there have been in addition to the almost endless works not mentioned on the relation of intervertebral disks to low back pain and sciatica investigators who have devoted themselves to studying the possible existence of another chiefly ligamentous etiology of the clinical sign. The present writer has endeavoured to make the most of his material in this respect as well and in what follows record the procedure adopted in the investigation and the results achieved.

The first section comprises a comparison between the incidence of changes in the three lower interspinous ligaments in the control group and the cases with back pain. It was felt that a difference one way or the other between the groups in the incidence of ligament changes could provide an answer to the question of ligament damage being the causative agent of pain in the lumbar spine.

The mean age was considerably higher in the cases with back pain. This was due to the difficulty of finding older patients who had never had this symptom. In order to improve the comparability of the series only half the total of the 41-50 year group cases with back pain was taken into consideration which means that the number of ligamentous changes was correspondingly reduced statistically. Occasional cases of a higher age were excluded in both groups.

|                      | Total number<br>of cases | Cases with<br>ligamentous<br>change |    |
|----------------------|--------------------------|-------------------------------------|----|
| Control cases        | 39                       | 11                                  | 28 |
| Cases with back pain | 178                      | 88                                  | 90 |

Table 15. Comparison between the incidence of ligament change in the control and the cases with back pain by age groups of proportional size.

The incidence of changes in the three lower lumbar interspinous ligaments shows a certain difference between cases with and without back pain but the difference is not marked. The number of cases with ligamentous changes but without back pain is however surprisingly large. The simple fact that there are changes in the interspinous ligaments in the cases with back pain is consequently no indicator of the ligamentous character of the pain. At the same time this would be expected only in a small number of such cases. A typical example of freedom from symptoms despite disseminated ligamentous changes is seen in Fig. 26. The patient was a tall man of 24 who was a prominent athlete and a decathlon competitor. He was extremely agile and had an unusually flexible back and to assume ligament ruptures would occur was consequently not unreasonable. Yet he had never had any symptoms referable to the back. *There is thus no chance of showing a connection between low back pain and changes in the interspinous ligaments by purely roentgenologic means.*

It is nevertheless worth while considering the following clinical roentgenologic approach. KEIL (1939) found that interspinous ligaments



Fig. 6a) Complete rupture. A prominent athlete of 40 with very little back trouble. Ligament rupture in the L3-4 and L5-S1 interspace. No relief for

give rise to referred pain on intraligamentous injections of saline. This ligament pain is identical in character with the pain produced from muscle—a continuous ache felt deep in the limb and trunk. The pain was segmental in nature and constant in its radius of distribution in repeated experiments. When the ligament between the L4-5 spinous process was injected the pain was distributed over the buttock to the outer and anterior part of the thigh and when the ligament between L5 and S1 spinous processes was injected the pain was felt from the buttock and outer side of the thigh and calf to the foot.

It was observed by BAATHUP (1940) in many experiments in patients with spinous process lumbago that an injection of novocaine between the processes would stop an acute or chronic attack in the course of a few hours while some patients with injections of the drug into the muscles where the pain was felt produced no relief. The elevation and the good result of some operations on spinous process in cases of chronic lumbago with complete

or partial resection of the processes seemed to BAASTRUP to indicate that the cause of pain was to be found in the processes or in the tissue between them. FRANK too emphasized the result of novocaine injections and operated only in cases in which the injection had had a convincing effect in the form of cessation of the pain. FAULSTICH & ABRAHAM on the other hand did not place particularly great reliance on such injections and pointed out that the incorrect deposition of the fluid or its faulty diffusion could lead to misinterpretations.

A sensation of pain arising from pressure over the interspinous interspace, so called trigger point pain, constituted according to HACKERT a strong indication of ligament relaxation and hence a pathologic ligament finding and was suggestive of an etiologic connection between these changes and low back pain. He considered that he could confirm the diagnosis by means of a temporarily pain killing local injection of an anaesthetic into the ligament. He treated a very large series of cases of this type with sclerosing injections and a curative effect was achieved in 82 per cent of them.

Twenty three patients with lumbar symptoms which had earlier resisted therapy were treated by HVID and modum HACKERT. Immediate and satisfactory relief of pain was produced in 17 cases by a tetrocaine injection into the interspinous ligament which was tender on palpation and considered to be the source of the pain. He achieved an excellent or good result for up to 2 years in 14 (82 per cent) of the cases. Sclerosing injections had no effect in 5 of the 6 cases which obtained no relief from pain from the anaesthetic injections.

In the majority of the present author's examinations of ligaments, pain was registered on palpation over the three lower interspinous spaces; this was not only at the beginning of the series. No pain was elicited in 133 cases and 67 of these had normal ligaments. Sixty cases had pain on palpation over one or several interspinous spaces and in this group only 19 cases had normal ligaments. Thus if there was no pain on palpation the interspinous ligaments were normal in every other case, but if the contrary was true only in barely every third case. These 60 cases with a pain response to palpation exhibited changes in 62 ligaments whereas the 133 cases without pain on palpation had change in 93 ligaments. Each comparison thus shows that *there is a certain connection between pain on palpation and changes in the lumbar interspinous ligaments*.

There is a very striking point which merits special emphasis. The 40 controls, none of whom had ever had any back pain, included only 3 patients who complained of pain on palpation over 4 interspinous spaces.

In spite of the small frequency of tenderness to palpation in the material included 22 cases with roentgenologically verified changes in the inter spinous ligaments. The corresponding figures for the back pain were 100, 57 and 91. Against the absence of tenderness to palpation among the 40 controls there are 57 cases with tenderness to palpation among the 100 cases with back pain. This very marked difference in the ratio favours the assumption that pain on palpation and back pain may have a common denominator, an etiologic connection, i.e. that the back pain in such cases arises from the region of the spinous process.

A note was made in 46 of the cases as to whether anaesthesia prior to the injection of contrast medium eliminated the tenderness to palpation of the interspinous space. This was found to be so in 11 cases, leaving only 3 cases that failed to respond in this way. Thirteen of the cases that did respond were perfectly normal both roentgenologically and at operation while only 2 of the 5 unresponsive cases were normal. The elimination of tenderness to palpation by injections of an anesthetic agent before a ligamentography is thus no greater aid in differentiation between the presence of pathologic inter spinous ligaments or their connection with back pain than the demonstration of tenderness on palpation. This does not rule out the possibility that intraligamentous injection may perhaps give more reliable information.

## DISCUSSION

The very small incidence of pain on palpation in the control series constitutes strong evidence of a connection between pain on palpation and the back pain which was demonstrated in the rest of the series. Its localization peaks at the same time in favour of the assumption that the pain arises from the spinous region. When such pain was elicited over one of the three lower interspinous spaces in about 50 per cent of the cases the underlying ligament was diseased, in 30 per cent of cases the underlying ligament was normal but an adjacent ligament pathologic and in 20 per cent of cases the three lower interspinous ligaments were normal both at operation and roentgenologically.

Pain on palpation may thus be regarded as a more reliable indication that back pain arises from the region of the spinous process than operative or roentgenologic demonstration of change in the inter spinous ligaments. It registers changes over a wider area and the pain provoking pathologic process can be located in a tissue contiguous to the inter spinous ligament.

One example of this is the area of insertion of the long dorsal muscles in the spinous processes

It was consequently impossible to find a completely reliable method for diagnosing pain referable to the spinal region. The evidence of pain in response to palpation over the spinous interspaces however is probably a fairly reliable diagnostic aid. It may be concluded that in about half these cases a pathologic interspinous ligament is the cause of the pain; in other cases it probably arises from some adjacent tissue. *Ligamentography may be a differential diagnostic aid in cases of pain on palpation. Negative roentgenologic findings rule out a ligamentous mechanism of pain production with fair certainty. When however the roentgenologic findings are positive a ligamentous etiology of the pain is probable.*

## IX. SUMMARY AND CONCLUSIONS

This investigation was prompted by observation made in connection with disk operations. KALLIO frequently encountered herniated ligaments in the two lower spinous interspaces while eliminating disk prolapse in the corresponding intervertebral space. The aforementioned changes were assumed to be of both theoretical and practical clinical significance in regard to the production of prolapse and low back pain.

THEO FRANK SÄLÄN to embark on his comprehensive examination of the lumbar supraspinous and interspinous ligament of an autopsy material. The present author evolved a roentgenologic method for the demonstration of interspinous ligaments and the changes detectable in them. This method is reviewed and the roentgenologic findings in the different type of changes in the interspinous ligaments are analyzed. The relationship between the change and various anatomic and pathologic circumstances are considered.

The series consisted of 229 cases, of which 153 had low back pain or sciatica. 50 with no history of back pain served as control. It was not until the 50—60 year age group was reached that a distinct increase in the incidence of pathologic ligaments with advancing age was observed. Men displayed a higher incidence of ligamentous change than women. Change in the lumbo-sacral ligament were most common in the control, while the L 4—5 ligament preponderated in the cases with back pain.

The posture of the lumbar spine appeared to affect the occurrence of change in the three lower interspinous ligament. As has been pointed out also by earlier investigators, changes were most common in the presence of marked lumbar lordosis and the same was the case when the patient was capable of extending the lumbar spine vigorously. On the other hand the origination of ligament changes was not due to the height of the spinous process. Interspinous ligaments attached to displaced spinous process were extremely frequently the sites of cavitation and rupture.

The minimum thickness of an interspinous ligament measured in the roentgenogram amounted to 4 mm. Ligament over 1 cm in thickness however were also fairly common. The L 3—4 ligament was thinner on the average than the ligaments closest to it caudally. Cavitation was most common in the thinnest ligaments in the L 3—4 interspace whereas it was noticeably uniform in ligaments of differing thickness in the L 4—5 interspace.

Cystic partial ruptures complete ruptures and slack ligaments were the principal pathologic types that could be diagnosed roentgenologically and whose roentgenologic characteristics could be described in detail. These various types of changes differed little from one another and to some extent occurred concomitantly in the ligaments. The result may be the cause of difficulties in roentgenologic interpretation in either case but they appear to have no practical significance.

No correlation could be shown between disk degeneration and the changes in the interspinous ligaments which also are of a degenerative character. Thus, these degenerative changes apparently have a divergent etiology. Prolapse of a disk and change in the interspinous ligament at the same level were established in 10 per cent of the prolapses. This incidence is of the same magnitude as prolapse and a normal ligament or prolapse and a changed heterologous ligament and consequently does not support the earlier hypothesis of a causal connection between prolapse and pathologic ligament. It was possible on the other hand to demonstrate a certain connection between trauma of the lumbar spine and changes in the three lower interspinous ligaments.

Finally the relationship between the changes in the three lower interspinous ligaments and low back pain was analysed. It revealed that after the control series and the cases with back pain had been adjusted to achieve comparability as regards age the incidence of pathologic ligaments was higher in the latter group. However every other symptom free back was the site of ligamentous changes, evidence that a positive roentgenologic diagnosis and low back pain cannot be equated.

Tenderness on palpation over the three lower interspinous ligaments was elicited very rarely in the controls but every third case with back pain reported painful sensations in response to palpation of an interspinous space, the underlying ligament was pathologic in half these cases. Tenderness to palpation therefore appears to be an indication of low back pain arising from the spinous processes. Pain was assumed to emanate e.g. from the sites of insertion of the long dorsal muscles in the spinous processes but its interspinous ligamentous etiology also seemed to be a factor to be considered. The significance of negative ligamentography in the differential diagnosis is emphasized and a certain clinical importance is accorded to positive roentgenologic findings.

## ACKNOWLEDGEMENTS

The present work was supported by grants from the Sigrid Juselius Foundation to which my very best thanks are due.



## REFERENCES

- BAASSTUJ ( I On the spinous processes of the lumbar vertebrae and the soft tissues between them and on pathological changes in that region *Acta radiol* 14 (1933) 3
- Proc spin vert lumb und einige zwischen diesen liegende Gelenkbildungen mit pathologischen Prozessen in dieser Region Fort in Rontgenstr 48 (1933 b) 430
- The diagnosis and roentgen treatment of certain forms of lumbago *Acta radiol* 21 (1940) 151
- BJORKROTH I Die subcutane Ruptur der distalen Sehne des Musculus bicip brachii *Acta chir Scandinav* 81 (1943) 30
- BRILLSFORD J Deformities of the lumbosacral region of the spine *Brit J Surg* 16 (1928—29) 362
- BURKHARDT H Über Spondylitis (Spondylosis) deformans *Med Klin* 30 (1934) 1349
- COMROE B I Arthritis Ilea and Iliacus 1933
- DAVIDSSON L Über die subcutanen Sehnenrupturen und die Regeneration der Sehne *Ann chir et gynaeec Iennise* 45 Suppl 6 (1936)
- FAULONC L LECHE I & AHRAN A La lombalgie par neurotose interepineuse *Iresse med* (1949) 34
- FICK R Handbuch der Anatomie und Mechanik der Gelenke In Handbuch der Anatomie des Menschen ed by Bardeleben Band II I Teil I Fischer Jena (1904)
- FLOYD W F & SILVER I H S Junction of erectors spinae in flexion of the trunk *Lancet* 260 (1951) 133
- FRANCK S Surgical treatment of interspinal osteoarthritis (skissing spine) *Acta orthop Scandinav* 14 (1943) 17
- FRIBERG S & HIRSCH C Anatomical and clinical studies on lumbar disc degeneration *Acta orthop Scandinav* 13 (1944) 222
- HACKETT G S Joint ligament relaxation treated by fibro osseous proliferation II Edit Charles C Thomas Springfield 1957
- JOSEPHANS G Kreuzschmerzen bei Dornfortsatzveränderungen *Ztschr Rheumaforsch* 13 (1954) 361
- KALLIO K E Syndrome of ruptured inter spinous ligament and its repair by use of free skin graft *J Bone & Joint Surg* 42—B (1960) 403
- Rupture of the lumbar interspinous ligament diagnosed by contrast medium x ray and reconstructed by free cutis graft *Bulletin of the hospital for joint diseases* vol 21 No 2 198
- KELLGREN J H On the distribution of pain arising from deep somatic structures (*Ann Sc* 1 (1933) 3)







FROM THE DEPARTMENT OF SURGERY (DIRECTOR PROF N OBEL)  
ROYAL VETERINARY COLLEGE STOCKHOLM SWEDEN

# INFLUENCE OF EXTENSIVE LAMINECTOMY ON THE SHAPE OF THE SPINAL CANAL

B FUNKOUIST and B SCHANTZ

LIBRARY  
The University of  
24/6/2024

COPENHAGEN 1962



|  |    |
|--|----|
| B Laminectomy with Retention of the Outer Compact Bone of the<br>Dorso-lateral Portion of the Arch | 42 |
| Material and Method  | 42 |
| Results  | 42 |
| Discussion   | 47 |
| Summary  | 49 |
| References   | 50 |

# I Laminectomy without Primary Narrowing of the Spinal Canal

## INTRODUCTION

Although complications fully compatible with the suspicion of spinal cord compression have been observed during the course of healing after extensive laminectomy (von Colmar 1954 Bette 1955 Behrend 1957 Pouyanne 1957) no detailed experimental investigation into the effect of that operation on the shape and volume of the spinal canal has heretofore been reported. While studying the therapeutic value of decompressive laminectomy for herniated intervertebral disks in the thoraco-lumbar region in the dog we made certain observations which suggested that the spinal canal following extensive laminectomy could be so narrowed as to occasion compression of the cord. We found these observations so interesting that we resolved upon an experimental study of this particular problem. Since the initial findings indicated that scar shrinkage of the granulation tissue bridging the surgical defect was a determinant factor in the spinal canal volume the experimental conditions were varied primarily with a view to preventing such shrinkage and its sequelae. Contracture of the surgically mobilized portions of the lumbodorsal fascia could not be excluded as a significant factor and hence the experimental conditions were also varied with respect to the treatment of the fascia. The first section of this investigation (I) is concerned with the effect in dogs subjected to laminectomy alone (with or without the secondary measure defined under subheadings A1 A3). The second section (II) deals with the effect in dogs whose spinal canals were narrowed at the start of healing by an epidural injection of paraffin given either before or immediately after the laminectomy.

### *Experimental Techniques Common to both Groups of Experiments (A and B)*

A total of 33 dogs ranging in age from 6 months to 2 years and in weight from 6 to 20 kg (mean 11 kg) were used for the experiments.

Anesthesia was induced by intravenous injection of Intraval® and maintained by a 4:1 mixture of nitrous oxide and oxygen. Muscle relaxation when indicated was produced by repeated intravenous injections of succinylcholine iodide.

|   |    |
|---|----|
| Gamma Laminatectomy with Retention of the Outer Compact Bone of the |    |
| Dorso lateral Portion of the Arch                                   | 42 |
| Material and Method   | 42 |
| Results   | 42 |
| Discussion  | 47 |
| References  | 49 |
|   | 50 |



# I Laminectomy without Primary Narrowing of the Spinal Canal

## INTRODUCTION

Although complications fully compatible with the suspicion of spinal cord compression have been observed during the course of healing after extensive laminectomy (von Colmar 1954 Bette 1955 Behrend 1957 Pouyanne 1957) no detailed experimental investigation into the effect of that operation on the shape and volume of the spinal canal has heretofore been reported. While studying the therapeutic value of decompressive laminectomy for herniated intervertebral disks in the thoraco-lumbar region in the dog we made certain observations which suggested that the spinal canal following extensive laminectomy could be so narrowed as to occasion compression of the cord. We found these observations so interesting that we resolved upon an experimental study of this particular problem. Since the initial findings indicated that scar shrinkage of the granulation tissue bridging the surgical defect was a determinant factor in the spinal canal volume the experimental conditions were varied primarily with a view to preventing such shrinkage and its sequelae. Contracture of the surgically mobilized portions of the lumbodorsal fascia could not be excluded as a significant factor and hence the experimental conditions were also varied with respect to the treatment of the fascia. The first section of this investigation (I) is concerned with the effect in dogs subjected to laminectomy alone (with or without the secondary measures defined under subheadings A1 A3). The second section (II) deals with the effect in dogs whose spinal canals were narrowed at the start of healing by an epidural injection of paraffin given either before or immediately after the laminectomy.

### *Experimental Techniques Common to both Groups of Experiments (A and B)*

A total of 33 dogs ranging in age from 6 months to 2 years and in weight from 6 to 20 kg (mean 11 kg) were used for the experiments.

Anesthesia was induced by intravenous injection of Intraval<sup>®</sup> and maintained by a 4:1 mixture of nitrous oxide and oxygen. Muscle relaxation was maintained by repeated intravenous injections of succinylcholine.

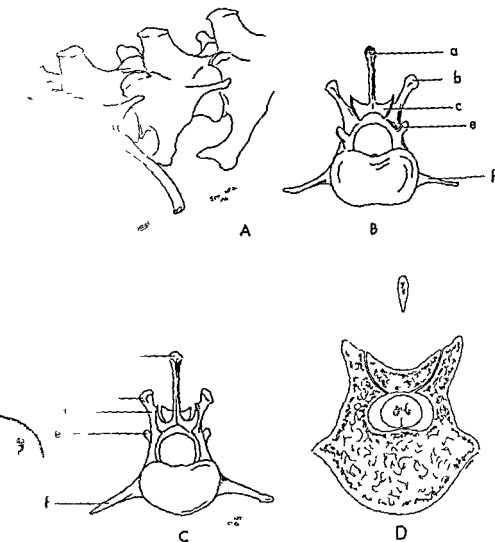


Fig 1 The anatomy of the dog's vertebral column in the region of the experimental operations

A) The vertebral column of the dog viewed obliquely from above at the junction between the thoracic and lumbar regions

B) First lumbar vertebra posterior view

C) First lumbar vertebra anterior view a) Spinous process b) Mammillary process c) Caudal (inner) articular process d) Cranial (outer) articular process e) Acetabulum f) Transverse process

D) Cross section of the first lumbar vertebra close to the disk of T13/L1 with the cord fixed in situ

(Celocurin®) in doses of 0.5 mg per kg body weight (Hansson 1958). Artificial respiration if needed during muscle relaxation was given with the aid of a "pulumat" (Dräger). The anatomy of the dog is such (fig. 1) that the present experimental surgery traumatized the spinal column and especially the articular processes to a much greater degree than would corresponding operative measures in humans. Following laminectomy the dogs were kept under observation for periods that will be indicated in the reports on the respective groups (usually at least 3 months). During the first two months or so of the observation period roentgenologic examination (lateral projection) was carried out, as a rule at intervals of approximately two weeks and subsequently about every four weeks. Some time during the last week of observation (generally a few days prior to sacrifice) the majority of the dogs were subjected to subarachnoid myelography with Kontrast L 3 with a technique which has been described elsewhere (Funkquist 1962).

At the end of observation the animals were sacrificed by rapid intravenous injection of Mebumal® in a dose of approximately 60 mg per kg body weight. Fixation was done immediately after death by intra arterial injection of 10 per cent formalin (dissolved in physiologic saline) with a previously described technique (Funkquist & Obel 1961). After about 24 hours storage of the cadavers in a refrigerator the relevant portion of the vertebral column was isolated and the bulk of the adjacent soft tissues dissected away. The excised specimens were immersed in formalin for approximately one week then frozen at  $-20^{\circ}\text{C}$  for 2-3 days after which they were sawn into sections about half the length of a vertebra. The sections were laid in the transverse plane—alternately through or in the vicinity of the disks and through the midpart of the vertebral body. These transverse sections with the spinal cord *in situ* were inspected in frozen condition, primarily with respect to the cross-sectional shape of the cord and the appearance of the tissue bridging the defect in the dorsal wall of the spinal canal (hereinafter called the defect). With the guidance of this preliminary inspection a number of representative specimens were selected from which frozen microsections 50-75  $\mu$  thick were prepared *ad modum* Lillberg (1958). The microsections were stained by Mayer's hemalum and eosin method. Immediately after sectioning the surface from which the microsections had been taken was photographed in frozen condition. In at least one animal from each group a sample of the tissue which had bridged the defect was taken from one of the transverse sections from the middle of the operative field. Following decalcification with nitric acid the sample was embedded in paraffin then sliced into sections approximately 6-7  $\mu$  thick and stained with hematoxylin and eosin.

### *Nomenclature*

In some of the dogs the operation produced dorso ventral flattening of the spinal cord as well as symptoms of motor loss. To facilitate classification of the experimental results the following grading of these two phenomena was employed

#### Dorso ventral Flattening of the Spinal Cord

The degree of flattening was determined in that transverse section which had shown the most conspicuous changes—usually a section from the middle of the operative field. Grading was based on the quotient of the vertical (V) and horizontal (H) axis of the cross section of the spinal cord as follows

- Grade I The transverse section exhibited distinct dorso ventral flattening, V/H however being equal to or greater than 0.60
- Grade II V/H was less than 0.60 but equal to or greater than 0.45
- Grade III V/H was less than 0.45 (lowest value recorded 0.30)

#### Symptoms of Motor Loss

- Grade I Mild paresis of the hind legs. These dogs walked without difficulty but tended to drag the dorsal surface of the hind paws along the ground. When moving briskly their hind quarters sometimes fell over particularly when the dogs made sharp turns.
- Grade II Moderate paresis of the hind legs. The dogs in this subgroup walked unsteadily and with great difficulty. It was difficult for them to advance the legs and they frequently set the dorsal surface of the hind paws on the ground.
- Grade III Subtotal to total paralysis of the hind legs. Walking was impossible, though some of the dogs showed a certain degree of voluntary motor activity of the hind legs.

## A Laminectomy with Complete Removal of the Dorsal and Dorso lateral Parts of the Arch, Including the Articular Processes

### MATERIAL AND METHOD

In a total of 27 dogs the dorsal and dorso lateral parts of the four arches in the region from T12 to L2 were excised by the following technique. The skin of that region was incised in the median line. The fascia was incised on each side of and contiguous to the spinous processes of T11 to L3. With the aid of scissors and a raspatory the muscles and periosteum were detached from the spinous processes as well as from the articular processes and the dorsal aspects of the arches. After removal of the spinous processes with bone cutting forceps the articular processes and the dorsal arches were gouged away sufficiently to expose the dorsal surface of the spinal cord—generally as far as a level between the dorsal tangent and the horizontal axis of the cross section of the spinal cord. The experimental conditions in group A were in other respects varied as follows.

1 In seven dogs the operation was confined to laminectomy as described above. In four of them the incision in the lumbo dorsal fascia was closed with continuous catgut no. 2 sutures and the skin incision with interrupted stainless wire sutures. In the remaining three the skin alone was sutured and the incision in the fascia left open. The observation time in this group was from 6 to 9 months for five dogs, 3 months for one, and 15 months for the seventh.

2 In six dogs the defect was filled with bone chips not exceeding 2 mm in diameter and having an average weight of approximately 3 mg (mean of 100). The chips were obtained by fragmenting with gouge forceps the excised parts of the arches. They were placed in two or three layers directly upon the dural surface between the cut surfaces of the arch. The fascial defect was sutured as above in three of these six animals and left open in the other three. Except for one dog with 4 months observation and another with 8½ months the observation time was 6-7 months.

3 The surface of the dura was in five dogs covered with a layer of subcutaneous fat fragments 2-4 mm in diameter obtained from the operative region. Above this fat layer were placed bone chips as described above. The fascia was sutured in two of the five animals and left open in the other three. The observation time ranged from 5 to 7 months.

4 In nine animals a strip of sheet polyethylene 0.02 mm thick was inserted

as to form a "septum" extending in the median plane throughout the surgical defect from the dura to the lumbodorsal fascia. Its ventral border was sutured with silk (no. 0) to the dorsal surface of the dura along a median line. In three of the animals subcutaneous fat plus bone chips and in another three bone chips alone were placed on the dura (on both sides of the plastic strip) as described in paragraphs 2 and 3 above. The dorsal edge of the plastic strip was, in all dogs, sutured to one edge of the fascial incision. Aside from this measure the fascia was not sutured. The observation time was 4-5 months, except for two animals in which it was approximately 3 months.

## RESULTS

A summary of the results is given in table I.

### *Groups 1-3*

*Functional disturbances* (motor loss and postural anomalies) — The seven dogs in which surgery had the character of laminectomy alone (group 1) tolerated the operation well; they exhibited no symptoms of motor loss during the first few weeks postoperatively. Paresis of the hind legs developed after some time in two cases (table I). The initial symptoms appeared, in one of these two, 2 months after the operation; then developed over the course of 2-3 weeks into moderate paresis (grade II) which persisted for at further week, whereafter the dog was sacrificed. In the second case mild paresis (grade I) was first detected one month after the operation and persisted about one month. For the remainder of the observation period (4 months) the motor activity was normal.

Kyphosis (of relatively mild degree) was observed in three dogs (table I). In two of them it was the sole symptom. The kyphosis persisted throughout the observation period in one of these two and was observed intermittently in the other. In the third dog which, one month after the operation, also presented symptoms of motor loss (described above) mild kyphosis was observed for the first 14 days postoperatively.

The eleven animals referable to groups (2 and 3) in which laminectomy was combined with transplantation of bone chips or of subcutaneous fat plus bone chips tolerated the operation itself satisfactorily, but diverged somewhat from the abovementioned dogs in regard to the postoperative course. Thus in four of the eleven paresis appeared in the first postoperative week. In one of these four in which paresis developed to grade II over the course of 8 days, the fascial sutures were removed on the eighth postoperative day and the tissues between fascia and dura were opened by a longitudinal incision extending the full length

Table 1

## Influence of Laminectomy on the Shape and Function of the Spinal Cord

(For grading of changes and symptoms see page 10 under the heading of Nomenclature)

| Section of<br>the<br>Spinal<br>Cord | Laminectomy   | Total<br>No. of<br>Dogs | Postural<br>Abnormalities |      | Symptoms of<br>Lumbosacral<br>Gingivitis |                        | Onset of Motor Loss<br>Symptoms |     |             |             | Shape of Cord<br>after the Spinal<br>Cord |                   | I |  |
|-------------------------------------|---|-------------------------|---------------------------|------|--|------------------------|---------------------------------|-----|-------------|-------------|---|-------------------|---|--|
|                                     |   |                         | Kyph                      | Lord | Symptoms                                 | Symptoms of Gingivitis |                                 |     | 1st<br>week | 2nd<br>week | 3rd<br>week                               | Nervous<br>System |   |  |
|                                     |   |                         |                           |      |  | I                      | II                              | III |             |             |   |                   |   |  |
| A                                   | A 1 Laminectomy with total excision of dorsal and dorsolateral parts of arch  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | With suture of lumbodorsal fascia   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 4   | 2                       | 3                         | 1    | 1  | 1                      | 1                               | 1   | 1           | 1           | 1   | 1                 | 3 |  |
|                                     | Without suture of lumbodorsal fascia  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 1                       | 2                         | 1    | 1  | 1                      | 1                               | 1   | 1           | 1           | 1   | 1                 | 3 |  |
|                                     | A 2 Laminectomy according to 1 Transplantation of autogenous bone chips to the defect   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | With suture of lumbodorsal fascia   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 2                       | 1                         | 2    | 1  | 2                      | 1                               | 2   | 1           | 1           | 1   | 1                 | 3 |  |
|                                     | Without suture of lumbodorsal fascia  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 3                       | 2                         | 2    | 1  | 1                      | 1                               | 1   | 1           | 1           | 1   | 1                 | 2 |  |
| A                                   | A 3 Laminectomy according to 1 Covering of dorsal surface of dura with autogenous fat thereafter transplantation of autogenous bone chips is in 2 |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | With suture of lumbodorsal fascia   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 2   | 2                       | 3                         | 1    | 1  | 1                      | 1                               | 1   | 1           | 1           | 1   | 1                 | 2 |  |
|                                     | Without suture of lumbodorsal fascia  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 3                       | 3                         | 3    | 1  | 1                      | 1                               | 1   | 1           | 1           | 1   | 1                 | 2 |  |
|                                     | A 4 Laminectomy as in 1 Median polyethylene strip in defect   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | Without suture transplantation  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 3                       | 3                         | 3    | 3  | 3                      | 3                               | 3   | 3           | 3           | 3   | 3                 | 3 |  |
|                                     | With transplantation of bone chips  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 1                       | 3                         | 3    | 3  | 3                      | 3                               | 3   | 3           | 3           | 3   | 3                 | 3 |  |
| B                                   | With transplantation of subcutaneous fat fragments and bone chips   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 2                       | 2                         | 2    | 2  | 2                      | 2                               | 2   | 2           | 2           | 2   | 2                 | 2 |  |
|                                     | Without suture of lumbodorsal fascia  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 3                       | 3                         | 3    | 3  | 3                      | 3                               | 3   | 3           | 3           | 3   | 3                 | 3 |  |
|                                     | Laminectomy with retention of dorsal part of portion of compact bone of arch  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | With suture of lumbodorsal fascia   |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 3                       | 3                         | 3    | 3  | 3                      | 3                               | 3   | 3           | 3           | 3   | 3                 | 3 |  |
|                                     | Without suture of lumbodorsal fascia  |                         |                           |      |  |                        |                                 |     |             |             |   |                   |   |  |
|                                     | 3   | 1                       | 3                         | 3    | 3  | 3                      | 3                               | 3   | 3           | 3           | 3   | 3                 | 3 |  |

a) At maximum intensity b) Transient c) Onset of fistula in surgical scar after 2 months

d) The fascial sutures removed on the eighth postoperative day e) Not comparable to other groups see text

of the operative field. The movements of the hind legs were dramatically improved immediately after this measure. They became quite normal within 48 hours and remained so for the rest of the observation period. Of the remaining three paretic animals which exhibited less severe symptoms (grade I), one showed a progressive improvement during the remainder of the observation period, one no change in the symptoms, and the third a gradual deterioration after about 3 months. A further one of these eleven dogs developed moderate paresis (grade II) somewhat later in the postoperative course—after approximately 2 weeks. Although this animal gradually improved, it still had mild paresis at the end of observation.

Kyphosis was observed in ten of the eleven dogs in group 2 and 3. It was most conspicuous in the five with motor loss in which it was present for the greater part of the observation period. Variations in the degree of kyphosis were generally correlated to variations in the intensity of paretic symptoms. In the other five dogs the kyphosis developed at varying intervals after the operation and was of short duration (as a rule 1-2 weeks).

When the incidence of paresis in all of the above groups (18 dogs) is considered in relation to the treatment of the fascia at operation, it emerges that paresis during the *first postoperative week* occurred in four of the nine animals in which the fascia had been sutured, but in none of the nine where the fascia had been left open. Further, this early paresis was limited strictly to dogs which had been subjected to transplantation of either bone chips or subcutaneous fat plus bone chips. Paresis of later onset (2 weeks, 1 month and 2 months respectively after operation) was noted in three animals—two in the group without fascial suture and one in the group with fascial suture.

*Roentgenologic changes* — Plain films (lateral projection) generally disclosed in the dogs which had undergone laminectomy alone no appreciable changes aside from the surgical defect in the spinal column (fig. 2) and in certain animals the aforementioned kyphosis which was also demonstrable on physical inspection. During the latter part of the observation period, however, some animals exhibited osteophytes simulating articular processes on those portions of the arches which adjoined the intervertebral disks.

When the operation had been supplemented by transplantation of bone chips or of subcutaneous fat plus bone chips, the contours of the chips were still discernible at the initial roentgen examinations. The chips progressively increased in size and decreased in number. Two to 4 months after the operation the graft had coalesced roentgenologically into a bone plate, though in most instances it was interrupted above the disks (fig. 3).

Mycelographic examination was undertaken in 18 animals. On lateral films





Fig 2 Radiograph (lateral exposure) of a preparation of a dog's vertebral column from T11 to L3. The animal was killed approx. 6 months after laminectomy on T12-L2 according to A.

No remarkable changes except the surgically produced defect in the vertebral column.



Fig 3 Radiograph (lateral exposure) of a preparation of a dog's vertebral column from T11 to L3. The animal was killed approx. 7 months after laminectomy on T12-L2 according to A, combined with transplantation of autogenous bone chips to the defect in the roof.

The grafts have fused to a bony plate forming a new roof of the spinal canal with the exception of the parts situated just above the disks.

of the operative field. The movements of the hind limbs improved immediately after this measure. They lasted 1 to 2 hours and remained so for the rest of the observation period. In three parietic animals which exhibited less severe paresis, a progressive improvement during the remaining 24-hour period, one no change in the symptoms and the third died after about 3 months. A further one of these eleven dogs developed paresis (grade II) somewhat later in the postoperative course—approximately 2 weeks. Although this animal gradually improved, it still has paresis at the end of observation.

Kyphosis was observed in ten of the eleven dogs in group 2 and was most conspicuous in the five with motor loss in which it was present for the greater part of the observation period. Variations in the degree of kyphosis were generally correlated to variations in the intensity of parietic symptoms. In the other five dogs the kyphosis developed at varying intervals after the operation and was of short duration (as a rule 1-2 weeks).

When the incidence of paresis in all of the above groups (18 dogs) is considered in relation to the treatment of the fascia at operation, it emerges that paresis during the *first postoperative week* occurred in four of the nine animals in which the fascia had been sutured but in none of the nine where the fascia had been left open. Further, this early paresis was limited strictly to dogs which had been subjected to transplantation of either bone chips or subcutaneous fat plus bone chips. Paresis of later onset (2 weeks, 1 month and 2 months respectively after operation) was noted in three animals—two in the group without fascial suture and one in the group with fascial suture.

**Roentgenologic changes** — Plain films (lateral projection) generally disclosed in the dogs which had undergone laminectomy alone no appreciable changes aside from the surgical defect in the spinal column (fig. 2) and in certain animals the aforementioned kyphosis which was also demonstrable on physical inspection. During the latter part of the observation period, however, some animals exhibited osteophytes simulating articular processes, on those portions of the arches which adjoined the intervertebral disks.

When the operation had been supplemented by transplantation of bone chips or of subcutaneous fat plus bone chips, the contours of the chips were still discernible at the initial roentgen examinations. The chips progressively increased in size and decreased in number. Two to 4 months after the operation the grafts had coalesced roentgenologically into a bone plate, though in most instances it was interrupted above the disks (fig. 3).

Myelographic examination was undertaken in 18 animals. On lateral films



Fig 5a Cross section through the middle part of L1. The animal was killed approx 3 months after laminectomy on T12 L2 according to technique A. The defect in the roof of the spinal canal is bridged by fibrous tissue which is attached to the fracture surfaces of the remaining portions of the arch. Severe dorso-ventral flattening of the spinal cord.

the observed myelographic changes appeared as filling defects in the dorsal subarachnoid space (fig 1a) and on frontal views as a broadening of the contrast column (fig 4b) and/or interruption of its borders. Myelographic changes were noted in 16 of the 18 dogs examined. In eight of them the changes were detectable on both lateral and frontal views, in five on lateral views only, and in three on frontal views only. As regards the type and degree of these changes no conspicuous differences were observed between the different groups. It should be noted, however, that both of the normal myelograms were referable to dogs which had been subjected neither to transplantation of tissue nor to suture of the fascia.

*Patho anatomic changes* — In those animals in which the operation had been confined to laminectomy without transplantation (group 1) the caudalmost and cranialmost parts of the defect (about half the length of a vertebra) were found to be bridged by osseous tissue. The rest of the defect was bridged with



Fig 5b Cross section close to the disk T13/L1 of the dog described in the legend to fig 5a

The defect is bridged by fibrous tissue situated at approximately the same level above the floor of the spinal canal as the corresponding tissue of the middle part of the vertebra (*cf* fig 5a) Severe dorso-ventral flattening of the cord Bilateral osseous processes (remnants of the articular processes or newly formed bone originating from such remnants) project dorsally to a level considerably higher than the above mentioned connective tissue membrane

fibrous tissue poor in cells In cross sections through the vertebrae situated near the middle of the operative field the lower border of this bridge of tissue largely coincided with a straight line between the upper edges of the remaining parts of the arch (fig 5a) In cross sections through or in the vicinity of the corresponding intervertebral disks the bridging fibrous tissue maintained the same level as in the middle of the vertebrae in spite of the fact that osseous processes emanating from the cranial or caudal ends of the vertebrae (remnants of articular processes or newformed bone *cf* roentgen findings) in some animals reached a higher level (fig 5b) In the cranial and caudal portions of the defect the bridge of fibrous tissue rose gradually to normal level The spinal cord in the middle of the region of laminectomy showed in each of the animals in this group dorso-ventral flattening of varying degree (fig 5 table 1), though invariably most pronounced above the intervertebral disks The deformation of the spinal cord was particularly marked in the dogs which had exhibited symptoms of motor loss

When bone chips or subcutaneous fat plus bone chips had been transplanted in conjunction with the laminectomy (groups 2 and 3) the defects were usually

bridged by osseous tissue over the midportions of the vertebrae. Above the disks, on the other hand, the bridge consisted of fibrous connective tissue in which ossified areas were interspersed. As in the group 1 animals, the ventral border of the tissue bridging the middle part of the defect was situated at the level of a line passing between the original cut surfaces of the arch. Cranially and caudally the bridge rose gradually to normal level. Dorso-ventral cord flattening of varying degree was noted in all cases in the central area of the operative field (fig 6). It was greatest, as in group 1, above the intervertebral disks. With a single exception—the aforementioned animal in which the fascial sutures had been opened—all dogs that had suffered from motor loss exhibited a high degree of cord deformation (grade III). In the exceptional case the cord was only slightly flattened (grade I).

#### *Group 4*

*Functional disturbances*—Each of the nine dogs in which a median polyethylene septum extending from the dura to the fascial incision had been inserted at operation showed normal motor activity during the first months. One of



Fig 6 Cross section through the disk T12/T13. The animal was killed approx. 5 months after laminectomy on T12 L2 according to technique A, which was combined with a transplantation of autogenous fat and bone chips to the defect in the roof of the spinal canal.

The defect is bridged by a continuous bone plate. There is a severe dorso-ventral flattening of the cord.



Fig 7 Cross section close to the disk T13/L1. The animal was killed approx 4 months after laminectomy according to A. The operation has been combined with autogenous transplantation of fat and bone chips after a strip of polyethylene had been placed in the median plane from the dorsal surface of the dura to the edge of the fascia incision. The defect in the roof is filled with fibrous connective tissue interspersed with ossified areas. Close to the median plane there is a vertical cleft extending down to the dorsal surface of the dura. Dorso-lateral flattening of the cord makes the cross section appear almost in the shape of a triangle.

the animals in which the insertion of this "septum" had been combined with transplantation of subcutaneous fat as well as bone chips suddenly developed subtotal paralysis (grade III) of the hind legs 2 1/2 months after the operation. The paralysis coincided with the onset of a fistula in the surgical scar and remained unchanged during the rest of the period of observation (7 days). The

remaining dogs displayed no motor disturbances during the observation period. One animal had mild kyphosis throughout the observation period and another showed transient moderate kyphosis during the first postoperative month. Lastly, the abovementioned dog with a fistula in the surgical scar had transient moderate kyphosis at the time of its onset.

*Roentgenologic changes*—Plain films (lateral projection) revealed changes essentially identical to those described for the corresponding animals in groups A1-3. Myelographic examination was carried out in seven of the nine dogs and disclosed, in six of them, dorsal filling defects of the character already described. The myelographic changes were located, as a rule, above the intervertebral disks in the central parts of the operative field. The seventh animal had a normal myelogram.

*Patho-anatomic changes*—The tissue bridging the defect was in principle of the same type as the one described for the corresponding experiments in groups A1-3. All animals had, in or near the median plane, a cleft which usually extended through the bridging tissue to a point contiguous to the dura. In two animals, however, some part of the clefts—located in one animal at T13 and in the other at the twentieth intervertebral disk—terminated 2 mm and 5 mm respectively above the dura. Although spinal cord deformation was noted in all animals, it differed from that in the previously described groups inasmuch as the flattening was principally dorso-lateral; the cord frequently having a triangular cross section (fig. 7).

## B Laminectomy with Retention of the Outer Compact Bone of the Dorso lateral Portion of the Arch

### MATERIAL AND METHOD

Six dogs were subjected to laminectomy, likewise from T12 to L2 inclusive, with the following technique. The procedure for incision of the skin and fascia, excision of the spinous processes, and detachment of muscle and periosteum from the dorsal aspects of the arches was as described in section A. By means of a pneumatic dental drill a median groove a few millimetres wide was made in the dorsal surface of the arches throughout the area relevant to the laminectomy (fig 8b and c). The turbine and hand grip for operating the drill were of Atlas Copco make (50 000 r.p.m.). Round burs with diameters varying from 1.1 to 2.9 mm were used. When the base of the groove had become so thin that it tended to sag under the burr, no further deepening was done, but excavation was continued in the lateral and latero-ventral directions (fig 8d). The dorso lateral portions of the arches were thus excavated by removal of the cancellous bone and the outer layers of the inner compact bone, while the outer compact bone was left intact. The outer (cranial) articular processes were largely undisturbed, but it was necessary to remove major portions of the inner (caudal) articular processes. Transection of the inner articular process

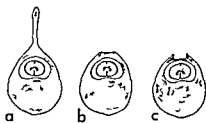


Fig 8 Outline drawings of the technique of laminectomy with retention of the outer compact bone of the dorso lateral portion of the arch (technique B)





was sometimes required particularly in small dogs and in these cases the isolated tip of the process was removed. Excavation of the arches was continued as far ventrad as the anatomy permitted—usually as far as a plane situated approximately midway between the dorsal tangent and the horizontal diameter of the spinal canal. The thin persistent remnant of the inner compact bone of the arch was then removed with a small gouge forceps. In the lateralmost portion it was difficult to reach the inner compact bone with this instrument without disturbing the spinal cord consequently a sharp bony ridge usually remained when the gouging had been completed (fig 8e). This ridge was removed with a small scoop. On termination of the above procedure all that remained of the dorsal wall of the spinal canal was a "shell" of outer compact bone in the dorso-lateral portion of the arch as well as the outer and sometimes, parts of the inner articular processes (fig 8f). In three of the animals the lumbodorsal fascia was sutured and in the other three it was left open. The skin in each case was sutured as described above. Except for one animal which was observed approximately 7 months the observation time was 4-5 months.

## RESULTS

A summary of the results is given in table I.

*Functional disturbances*—All dogs exhibited when they had recovered from the anesthesia normal motor activity until the end of the observation period (table 1). While no kyphosis was observed in this group moderate lordosis occurred in one dog (in which the fascial incision had not been sutured).

*Roentgenologic changes*—Plain films (lateral projection) revealed no changes of note aside from the surgical defect in the vertebral column (fig 9). Three animals were subjected to myelography and in two of them the contrast column on the frontal view was abnormally narrow (fig 10). One of these two also presented a slight impression in the dorsal contrast line above the disk between T13 and L1. An impression of the last mentioned type was noted as the only change in the third animal.

*Patho anatomic changes*—As in the previously described type of laminectomy the defect was bridged by osseous tissue in its caudalmost and cranialmost parts. In some animals osseous bridge were found also on vertebrae situated more centrally in the region of laminectomy. The remainder of the defect was filled with fibrous tissue poor in cells. The cross sections at the intervertebral disks showed in some animals on the inside of the remaining parts of the articular

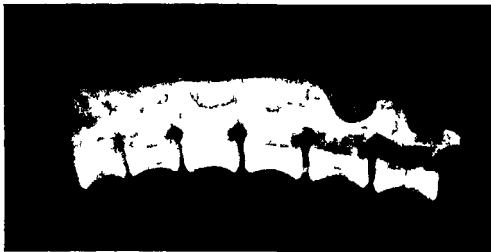


Fig 9 Radiograph (lateral exposure) of a preparation (T11 L3) of the vertebral column of a dog. The animal was sacrificed approx 5 months after laminectomy on T12 L2 according to B

There are no remarkable radiological changes beyond the defect in the vertebral column produced by surgery (including removal of the inner articular processes)

processes areas of new formed bone with interspersed islands of cartilage. In no case was there any demonstrable dorso ventral flattening of the cord such as that which followed type A laminectomy (fig 11). One of the animals showed, however, in the central area of the operative field, a certain degree of lateral flattening, the vertical axis of the cross section of the cord being equal to or somewhat greater than its horizontal axis (fig 12). The vertebral sinus in the last mentioned dog showed heavy filling (fig 12).

## DISCUSSION

In all dogs which had undergone laminectomy at the junction of the thoracic and lumbar regions with complete removal of the dorsal and dorso-lateral parts of the arch as well as the articular processes (type A laminectomy), dorso ventral flattening of the cord occurred during healing. It was possible to avoid this deformation of the cord following laminectomy in the same area, by conserving the outer compact bone in the dorso lateral part of the arch as well as the outer articular processes (type B laminectomy). By virtue of the special technique employed at the patho-anatomic examination those spinal cord deformations which followed type A laminectomy could be attributed to the fact that the lower border of the fibrous tissue which bridged the postlaminectomy

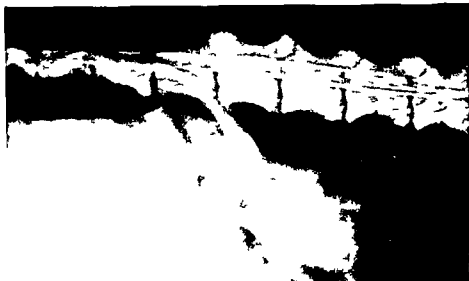


Fig 10 Myelogram from an experimental dog approx 4 months after laminectomy on T12 L2 according to B

a) Lat ral exposure Small dorsal impressions in the contrast column above the disks T13/L1 and L1/L2 (cf Fig 4a)



b) Ventro dorsal exposure Th contrast column is narrower than normally within the middle part of the laminectomized area

tomy defect largely coincided with a straight line between the cut surfaces of the remaining parts of the arch

The abovementioned variations in the shape of the cross section of the cord following different types of laminectomy might be accounted for as follows. If the granulation tissue bridging the post laminectomy defect is assumed to be fixed to both cut surfaces of the arch it may form on scar contracture a tense membrane between those two attachments. Following type A laminectomy at which the arch is excised down to a plane below the dorsal tangent of the cord, this membrane will then compress the cord against the base of the spinal canal. Following type B laminectomy which conserves the dorso lateral portion of the compact bone of the arch, the granulation tissue will have its attachments at a higher level and the membrane associated with scar contracture will form at a greater distance from the canal floor thus precluding spinal cord compression.

This tentative explanation of the pathogenesis of post laminectomy compression of the cord is supported by the observations referable to those dogs in



Fig 11 Cross section close to the disk T13/L1. The animal was destroyed approx 7 months after laminectomy on T12/L2 according to B. The defect in the roof of the spinal canal is filled with fibrous connective tissue. There is no obvious change of the shape of the cross section of the spinal cord.



Fig 12 Cross section close to the disk T12/T13. The animal was killed approx 5 months after laminectomy on T12 L2 according to B. The cord is flattened laterally. The vertebral sinuses are well filled.

which after laminectomy the space between the cord and the fascial incision was divided by a median "septum" of polyethylene. The application of this plastic strip was designed to avert the effect of the transverse component of the scar contracture. Although appreciable deformation of the cord occurred even in these animals, the space available to the spinal cord seemed to be considerably greater in the vicinity of the median plane so that the cord often assumed a triangular cross section. Also worthy of note is the absence of motor loss in these dogs with the exception of one in which a fistula opened in the surgical scar thus showing that inflammatory changes had probably contributed to the genesis of the symptoms. The fact that a certain degree of compression arose even though the tissue covering the cord had been interrupted in the median plane may well have been attributable to the longitudinal component of the shrinkage. Owing to the physiologic dorsal convexity of the vertebral column in the pertinent region even the longitudinal shrinkage probably tends to compress the cord against the canal floor.

It is evident from the foregoing that of the operative methods tried only that technique (B) which conserved the outer compact bone in the dorso lateral portion of the arch afforded an adequate safeguard against grave spinal cord compression following extensive laminectomy at the junction of the thoracic and lumbar segments of the vertebral column. Although slight lateral flattening was noted in one case after laminectomy of this type, it was not associated with neurologic symptoms. A probable explanation of the latter type of spinal cord deformation is that the scar shrinkage serves to raise the meninges—and with them the cord itself—towards the fibrous tissue bridge which unites the tips of remaining parts of the arch. Proliferative processes on the damaged inner surface of the arch may, of course, have contributed to the deformation but the presence of a distended venous sinus ventral to the cord (fig 12) lends weight to the assumption that the lateral flattening is largely attributable to elevation of the dura.

Our attempts to reduce the tendency towards stricture of the spinal canal by transplanting autogenous bone chips in order to promote early ossification of the tissue bridging the defect did not lead to the desired results. The same was true of the experiments designed to increase the diameter of the bony canal by clothing the dorsal surface of the dura with autogenous fat prior to the application of bone chips. The experiments may nevertheless be of some interest when considered in relation to the varied treatment of the lumbodorsal fascia. Paresis appearing within the first postoperative week and not attributable to the surgical trauma *per se* was observed only in cases where the fascia had been sutured in conjunction with transplantation of autogenous tissue. The dramatic improvement in the functional state of the spinal cord which in one of these animals, followed opening of the fascial suture and underlying tissue down to the dura suggests that an increase of pressure in that part of the surgical cavity which lies between the fascia and dura is a determinant factor in this early paresis. Since postoperative bleeding may well fill a volume equal to that occupied by the tissue grafts in these experiments trials of therapeutic laminectomy with omission of the fascial sutures seem worth while. It is worthy of note that in our experimental animals the relatively large wound cavity which is inevitable if the operation is performed in the latter way did not entail any serious disadvantages in regard to wound healing.

As regard the incidence of kyphosis during the postoperative course there is a substantial difference between the two operative methods. Kyphosis arising only after type A laminectomy, at which the dorsal portion of the arch as well as the articular processes were excised. The cause of the difference may be that the animals having lost the supportive action of the articular processes were compelled to hold their backs in a kyphotic posture to counteract the

effect of gravity. It is nevertheless conceivable that the spinal canal stricture was a contributory factor for the cross sectional area of the cord as demonstrated by Breig (1960), is reduced on bending of the spinal column. Possibly therefore the animals assumed a kyphotic posture to lessen the pain which could well have resulted from compression of the cord and the nerve roots by the walls of the narrowed spinal canal.

## SUMMARY

The healing process following thoraco-lumbar laminectomy comprising several vertebrae in dogs entails a risk of dorso ventral compression of the spinal cord when the arch including the articular processes is excised down to a horizontal line bisecting the spinal cord. The compression is apparently due to the fact that the granulation tissue which bridges the dorsal defect in the spinal canal tends on scar shrinkage to follow a straight line between the cut surfaces of the remaining parts of the arch thereby pressing the cord against the base of the spinal canal. Following laminectomy which conserves the outer portions of the arch including the outer articular process dorso ventral compression of the cord may be avoided—apparently because the scar tissue in this case will be attached to points well above the base of the spinal canal.

## REFERENCES

- Brend C M. Aus dem Gebiet der chirurgischen Erkrankungen des Rückenmarks und der Wirbelsäule. *Medizinische* 1 (1957) 565.
- Bite H. Folgezustände von Laminektomien an der Halswirbelsäule. *Zschr orthop* 85 (1955) 564.
- Breig A. *Biochemistry of the central nervous system*. Almqvist & Wiksell, Uppsala 1960.
- Colmar A. Die Wirbelsäuleninsuffizienz nach ausgedehnter Laminektomie. *Arch orthop Unfallchir* 46 (1954) 445.
- Funkquist B. Thoraco-lumbar myelography with water soluble contrast medium in dogs. *J small anim Pract* 3 (1962) 53.
- Funkquist B. *Ibid* 3 (1962) 67.
- Funkquist B & Obel V. Effect on the spinal cord of subarachnoid injection of water soluble contrast medium. *Acta radiol* 56 (1961) 449.
- Hansson C H. Anaesthesia in the dog using short acting barbiturate and nitrous oxide supplemented with succinylcholine iodide and positive negative artificial respiration. *Nord Vet Med* 10 (1958) 407.
- Poujanne L. Déformations du rachis consécutives aux laminectomies étendues. *Bordeaux Chir* (1957) 65.
- Ullberg S. Autoradiographic studies on the distribution of labelled drugs in the body. *Proceed of the sec. UN internat. conf. on the peaceful uses of atomic energy* (Geneve) 24 (1958) 248.

## II Laminectomy in Connection with Primary Experimental Narrowing of the Spinal Canal

### INTRODUCTION

The influence of extensive laminectomy on the shape of the spinal canal in healthy experimental dogs has been reported in an earlier paper (Part I). The results show that laminectomy in the region from T12 to L2 with complete removal of the dorsal and dorso-lateral parts of the vertebral arch including the articular processes down to a level below the dorsal tangent of the spinal cord (the A technique according to Part I) involves a major risk of dorso-ventral compression of the cord during healing. On the other hand, if the outer compact bone of the dorso-lateral parts of the arch including the outer articular processes are spared at laminectomy (the B technique) the abovementioned late complication can be prevented. In order to investigate how the process of healing after extensive laminectomy affected the spinal cord when the latter had been displaced in the dorsal direction—as may occur, for instance, in the case of a median herniated disk—the following experiments were carried out.

#### *Experimental Techniques Common to both Groups of Experiments (A and B)*

A total of 22 dogs ranging in age from 6 months to 2 years and in weight from 8 to 15 kg (mean 11.5 kg) were used for the experiments. Experimental conditions with regard to anesthesia and artificial respiration were the same as those described in Part I. Anesthesia was not combined with muscle relaxation in those experiments where transvertebral injections of paraffin were given.

After laminectomy the animals were observed for a period specified below in connection with the reports on the different experimental groups. At the end of the observation period the dogs were sacrificed and subjected to fixation by means of intra-arterial injections of formalin according to the method described in Part I. Patho-anatomic examination of the operative region was carried out with technique also described in Part I.

In order to elevate the spinal cord by introducing a paraffin mass into the ventral epidural space with one or the other of the following two methods



### *Trans vertebral Injection of Paraffin Wax*

An extensive thoracotomy was performed in the eighth intercostal space on the left side. The incision through the soft parts was extended ventrally over the median plane whereby the eighth sternebra became accessible from the ventral side. In order to widen the opening in the thorax, the seventh and eighth ribs with the intercostal muscle tissue were cut at the costochondral junction. The ventral side of T10 and the adjoining parts of T9 and T11 were exposed by blunt dissection after double ligation and division of the relevant intercostal vessels. In the caudal part of T10 a shallow excavation was made by means of a small gouge in the median ridge situated on the ventral surface of the vertebra. From this excavation a channel was bored through the main part of the body of the vertebra by means of a stainless steel pin. The pin the tip of which had been ground to a triangular pyramid had a length of 40 cm and a diameter of 3 mm. In order to ensure as far as possible that the bored channel would be located in the median plane the sternum was used as a directional device for the pin. Thus on inserting the pin into the thoracic cavity it was first passed through the eighth sternebra in the manner illustrated in fig. 1. The channel bored through the sternum and the vertebra was progressively widened by replacing the 3 mm pin with pins of respectively 4, 5 and 6 mm in diameter. All pins were provided with adjustable stop rings (fig. 1) which were set with respect to the vertical diameter of the body of the vertebra according to the roentgenograms. Finally the bored channel was extended into the spinal canal by means of a 6 mm pin with a chisel shaped tip. Since the bored channel had not formed a right angle to the spinal column but was directed obliquely

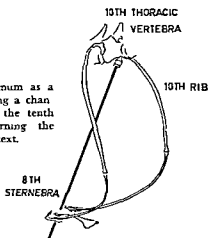


Fig 1 The use of the sternum as a directional device for boring a channel through the body of the tenth thoracic vertebra. Concerning the details please refer to the text.

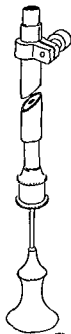


Fig 2a



Fig 2b

Fig 2 Elevation of the spinal cord by transvertebral injection of paraffin wax

- a) The trocar (fitted with outer tube and stop ring) for the injection of paraffin. Concerning the details please refer to the text  
 b) The instrument inserted in the channel through the body of the vertebra during the injection into the spinal canal of paraffin wax heated to a salve like consistency

caudalward, the chisel edge was bevelled to a corresponding angle so that the inner aperture should also encompass the caudal parts of the bored channel. The stop ring of the chisel shaped pin was repeatedly reset until it could be ascertained by probing that the entire circumference of the bored channel had reached the spinal canal. During the last stage of the procedure the boring was carried out with small oscillatory movements of the pin whereby it was continuously checked that the acute angle of the edge was directed caudalward. A trocar was used to introduce paraffin into the spinal canal (fig 2a). The trocar cannula had a caliber of 15 mm, an outer diameter of 4.9 mm and a length of approximately 80 mm. It was mounted for connection of an ordinary "Record" hypodermic syringe. The trocar was inserted into an outer tube (fig 2a) with a caliber of 5 mm and an outer diameter of 6 mm. The length of the outer tube was so adjusted that the orifice of the trocar cannula fell about 10 mm short of the orifice of the outer tube when the trocar was completely inserted. The outer tube was fitted with an adjustable stop ring which, with the guidance of the roentgenogram, was so adjusted that the mouth of the outer tube was positioned a few millimetres ventral to the opening of the bored channel in the spinal canal when the stop ring was in contact with ventral surface of the vertebra. Prior to insertion of the assembled instrument into the bored

channel the trocar and that part of the outer tube in front of the orifice of the trocar cannula were filled by a syringe with melted paraffin (Merck<sup>®</sup> melting point  $56^{\circ}$ – $58^{\circ}$  C) at a temperature of approximately  $80^{\circ}$  C. When the paraffin had cooled to a salve like consistency the instrument was inserted so far into the spinal canal that the stop ring came into contact with the ventral surface of the vertebra. The paraffin in the trocar cannula (approximately 0.1 ml) was thereafter pressed into the outer tube with the mandrin. A 1 ml tuberculin syringe was then used to inject 0.1 ml paraffin at about  $80^{\circ}$  C into the trocar. After cooling for about 10 seconds in the trocar cannula this paraffin was pressed into the outer tube with the mandrin. This procedure was repeated until the desired effect was obtained. As shown in fig. 2b the injected paraffin is successively pressed into the spinal canal in the form of a column of semi solid consistency. Where this transvertebral injection was given *before laminectomy* the introduction of paraffin was as a rule continued until the animal reacted with distinct (transient) spasm of the hind legs. On the basis of preliminary experiments on cadaver however the maximum dose was limited to 0.6–0.9 ml depending on the size of the animal and at this dose the injections were discontinued even if no spasm had occurred. Where the paraffin injection was given *after laminectomy* the introduction of paraffin was continued until the spinal cord showed a distinct dorsal bulge. After completion of the paraffin injection the apparatus was cooled for a few minutes with gauze soaked in physiological saline solution at room temperature. The apparatus was then removed. The thorax was closed with chromic catgut no. 2 sutures and the skin with stainless 0.21 mm wire sutures.

#### *Introduction of Paraffin Wax into the Spinal Canal via the Laminectomy Opening*

Laminectomy was performed on T12, T13, L1 and L2 with one of the two methods described below. A plastic tube was made by welding the edges of sheet polyethylene 0.02 mm thick; the tube had a caliber of approximately 5 mm and a length of about 15 mm. One end of this tube was ligated with 3/0 silk a few millimetres from the opening and at the other (free) end a suture (of the same material) with long ends was inserted. By means of doubled 0.5 mm wire the bent end of which was twisted into a loop the ends of the suture were inserted under the spinal cord at the level of the disk between T13 and L1 between the relevant nerve roots at an angle of approximately 45 degrees to the median plane. By gently pulling the suture the polyethylene tube was drawn so far under the cord that the aforementioned ligature reached the base of the spinal canal lateral to the cord. After removal of the tube a probe

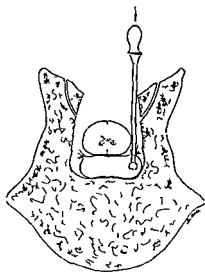


Fig 3 Elevation of the spinal cord and its meninges by injection (via the laminectomy opening) of paraffin wax into a polyethylene tube which is placed in the epidural space ventrally to the dura

pointed cannula was inserted so far into the free end of the tube that its bulb tip was positioned at the base of the spinal canal lateral to the spinal cord at the opposite side to the ligature. In this position it was fixed with a ligature drawn tightly around the polyethylene tube and the cannula just above the bulb tip of the latter (fig 3). Paraffin was then injected into the polyethylene tube as described earlier. The paraffin was injected at a temperature of about  $80^{\circ}\text{C}$  into the cannula in doses of 0.1 ml, was allowed to cool for about 10 seconds and was then pressed in semi solid condition into the polyethylene tube by a mandrin fitted into the cannula. The procedure was repeated until the spinal cord showed a distinct dorsal bulge. The requisite dose varied from 0.25 ml to 0.30 ml. After the paraffin had cooled for about 5 minutes the ligatures were removed and the polyethylene tube was fixed with a catgut suture (3/0 catgut) on each side to the musculature contiguous to the spinal column. The parts of the polyethylene tube outside the catgut sutures were thereafter cut off.

## A Laminectomy with Complete Removal of the Dorsal and Dorso lateral Parts of the Arch, Including the Articular Processes

### MATERIAL AND METHOD

Laminectomy was performed on a total of 11 dogs with the technique described in Part I, section A. The lumbodorsal fascia was not sutured. Prior to or in connection with the operation narrowing of the spinal canal was experimentally induced by one of the methods described above. The experimental conditions *within group A* were thus varied as follows:

1 Transvertebral paraffin injections at T10 were given in four dogs. When the animals had recovered 8 to 15 days after the first operation laminectomy on T9 to T12 was performed. The paraffin was not removed at operation. The observation period was approximately 10 weeks as from the date of laminectomy.

2 Laminectomy on T9 to T12 was performed in four dogs. Before the wound was closed a transvertebral paraffin injection was given at T10 with the previously described technique. The observation period was approximately 10 weeks.

3 Laminectomy on T12 to L2 was performed in three dogs. Prior to closure of the wound an epidural paraffin injection over the disk between T13 and L1 was given via the laminectomy opening with the technique described earlier. The observation period was four to five weeks.

### RESULTS

A summary of the results is given in table I.

With regard to the nomenclature used for the evaluation of symptoms of motor loss and degree of spinal cord compression see Part I page 10 under the subheading of "Nomenclature".

*Functional disturbances* — The postoperative course was as follows in the four animals described under the subheading of 1 that had received paraffin injections in the spinal canal at T10 level and in which laminectomy was performed 8 to 15 days later on T9 to T12 with complete removal of the dorsal and



Fig 4 Cross section through T10 close to the disk T9/T10 The dog was killed about 10 weeks after laminectomy on T9 T12 according to technique A

The defect in the roof of the spinal canal is bridged by a membrane of fibrous connective tissue which is fixed to the dorsal surface of the costal attachments or to the transverse processes i.e. to points which are situated rather high above the floor of the spinal canal No notable decrease of the vertical diameter of the canal

dorso lateral parts of the arches Following the paraffin injection symptoms of motor loss varying from moderate paresis to subtotal or total paralysis were present (grade II in one animal, and grade III in three animals one of which had spastic paralysis) The motor function gradually improved during the period prior to laminectomy At the time of operation one dog had mild paresis (I) two moderate paresis (II), and one paralysis (III) In one of the animals laminectomy did not affect the motor function In two of the dogs the operation caused a slight exacerbation of a few days duration In one case the existing paresis increased from grade II to grade III The exacerbation had a duration of approximately three weeks In this case wound infection with heavy suppuration occurred postoperatively Otherwise all the dogs showed gradual progressive improvement after the operation At the end of observation all the dogs were able to walk three had mild paresis (grade I) The fourth dog, in which spastic paralysis was present after the paraffin injection, had grade II paresis at the end of the observation period

Of the four animals described under the subheading of 2 that had received a transvertebral paraffin injection at T10 after excision of the dorsal and dorso lateral parts of the arches in the region T9 to T12 (with technique A) three



Fig 5 Cross-section through T10 Transvertebral injection of paraffin wax. Laminectomy on T9 T12 according to technique A Symptoms of motor loss which followed the paraffin injection (grade II) gradually improved during the period prior to laminectomy This caused a slight exacerbation of a short duration. After this the symptoms gradually improved until the end of the observation period (grade I) The animal was destroyed about 10 weeks after the laminectomy

The defect is bridged by a membrane of fibrous connective tissue which is fixed to the dorsal surface of the transverse processes To the left there is a paraffin mass displacing the cord to the right and causing a dorsal lateral impression of the cord's cross-section

showed a largely normal motor function when they had recovered from the anesthesia The condition of these dogs remained unchanged throughout the observation period The fourth dog had postoperatively moderate paresis (grade II) which gradually subsided to mild paresis (grade I) during the observation period

Of the three dogs described under the subheading of 3 where laminectomy had been performed on T12 to L2 and where paraffin had been introduced into the ventral epidural space via the incision made at laminectomy one showed mild and another moderate paresis (grade I and grade II respectively), while the third animal had a normal motor function The former two dogs showed a gradual motor improvement during the fortnight following the operation Approximately two weeks after the operation all three dogs had incipient motor impairment of the hind legs which gradually increased during the observation period At the time of sacrifice two of the animals had spastic paralysis (grade III) and the third subtotal paralysis (grade III) with some remaining voluntary motor function

Table I

Influence of Laminectomy in Animals with Experimental Narrowing of the Spinal Canal  
(For grading of changes and symptoms see part I page 10 under the heading of Nomenclature )

| Experiments | Experiment (Condition)   | Total Number of Dogs | Condition after Laminectomy |        |         |                                 |    |     | Condition at End of Observation |                |                                 |    |     |                | Shape of Cross-section of the Spinal canal |                                   |  |  |  |  |
|-------------|--|----------------------|-----------------------------|--------|---------|---------------------------------|----|-----|---------------------------------|----------------|---------------------------------|----|-----|----------------|--|-----------------------------------|--|--|--|--|
|             |  |                      | No. of Animals              |        |         | Symptoms of Motor Loss of Grade |    |     | No. of Animals                  | Motor Activity | Symptoms of Motor Loss of Grade |    |     | No. of Animals | Flattening                                 | Dorso-ventral Flattening of Grade |  |  |  |  |
|             |  |                      | No. I                       | No. II | No. III | I                               | II | III |                                 |                | I                               | II | III |                |  |                                   |  |  |  |  |
|             |  |                      |                             |        |         |                                 |    |     |                                 |                |                                 |    |     |                |  |                                   |  |  |  |  |
| A1          | Transvertebral paraffin injection at T10 Type A laminectomy of T9 T12 8 to 15 days after injection | 4                    |                             | 1      | 1       | 2                               |    |     |                                 | 3              | 1                               | -  | -   | -              |  |                                   |  |  |  |  |
| A2          | Type A laminectomy of T9 T12 with transvertebral paraffin injection at T10                         | 4                    | 3                           | 1      |         |                                 |    |     | 3                               | 1              |                                 | -  | -   | 2              |  |                                   |  |  |  |  |
| A3          | Type A laminectomy of T12 L2 with paraffin injection over T13 L1 via the laminectomy opening       | 3                    | 1                           | 1      | 1       |                                 |    |     |                                 |                |                                 |    |     | 3              |  |                                   |  |  |  |  |
| B1          | Transvertebral paraffin injection at T10 Type B laminectomy of T9 T12 9 to 17 days thereafter      | 4                    | 1                           | 2      | 1       |                                 |    |     | 1                               | 3              |                                 | -  | -   | -              |  |                                   |  |  |  |  |
| B2          | Type B laminectomy of T9 T12 with transvertebral paraffin injection at T10                         | 4                    | 2                           | 1      | 1       |                                 |    |     | 3                               | 1              |                                 | 3  | -   | -              |  |                                   |  |  |  |  |
| B3          | Type B laminectomy of T12 L2 with paraffin injection over T13/L1 via the laminectomy opening       | 3                    | 1                           | 2      |         |                                 |    |     | 2                               | 1              |                                 |    |     | 3              |  |                                   |  |  |  |  |

a) In the animals in these groups the dorso-ventral flattening of the spinal cord could not be graded owing to deformation caused by the lateral position of the paraffin

b) In two animals (A7) and one animal (B2) respectively grading could not be done for reasons mentioned under a





Fig 6 Cross section close to the disk T10/T11 Laminectomy on T9/T12 according to technique A with simultaneous elevation of the spinal cord by means of a transvertebral injection of paraffin wax. The symptoms of motor loss (grade II) exhibited by the animal on awakening gradually diminished and persisted at the end of the observation time only as a slight paresis (grade I). The animal was killed about 10 weeks after the laminectomy.

The defect in the roof of the spinal canal is bridged by a membrane of fibrous connective tissue which is fixed to the dorsal surface of the transverse processes. The spinal cord and its meninges are elevated by a ventral paraffin mass to such a degree that the distance between the ventral surface of the dura and the bottom of the canal is about one normal cord diameter. Moderate (grade II) dorsoventral flattening of the cord.

*Patho anatomic changes*—Postoperative behaviour of the spinal column was similar to that described under A in Part I. The cranialmost and caudalmost parts of the defect made by laminectomy (approximately one half the length of the vertebra) were thus bridged by bone, while the remainder was filled with fibrous tissue with few cells. In those animals described under the sub-headings of 1 and 2 in which the operation was performed within the region of T9 and T12 this tissue membrane which seemed to a large extent to be fixed to the dorsal surface of the costal attachments or to the transverse processes (fig. 4) was situated at a greater distance from the base of the spinal canal.



Fig 9 Cross-section close to the disk L1/L2 Laminectomy according to technique B with simultaneous elevation of the cord as described under 7 After recovery from the anaesthesia the dog showed almost normal movements which persisted to the end of the observation time The animal was destroyed about 5 weeks after the operation

The defect in the roof is bridged by fibrous connective tissue Ventrally in the epidural space there is a paraffin mass that elevates the cord and its meninges to such an extent that the distance between the ventral surface of the dura and the bottom of the spinal canal corresponds to approx 1 normal cord diameter There are no signs of cord deformation caused by shrinkage or proliferation of tissue

(grade II) but were able to walk These two animals showed progressive improvement At the end of the observation period two animals showed normal function, and signs of mild paresis were present in the third

*Patho anatomic changes* —The reaction of the spinal column was largely identical with that described for the corresponding operations in Part I In this series too some animals showed considerable bone and cartilage production from the medial surface of the remaining parts of the articular processes

Irrespective of the influence of paraffin injections the effect of the operations on the cross section of the spinal cord was also the same in principle as



Fig 10 Cross-section through the disk T13/L1 of the animal which has been described under fig 9

Ample formation of bone and cartilage on the inside of the remaining parts of the articular processes. The remainder of the defect is filled with fibrous tissue. The cord and its meninges are elevated to such an extent that the distance between the ventral surface of the dura and the bottom of the canal corresponds to approx  $\frac{3}{4}$  of a normal cord diameter. In the median plane and to the left there is ample space vertically for the cord but to the right there is a dorso-lateral flattening of the cord section corresponding to the above mentioned proliferation of bone and cartilage.

that reported in Part I under B 1 c there was no considerable dorso-ventral compression of the cord but in some animals a tendency to lateral flattening. Those changes of the shape of the cord which are referable—directly or indirectly—to the injection of paraffin are described below.

In the majority of those animals (groups 1 and 2) that had received transvertebral paraffin injections the paraffin had passed to the side of the spinal cord and displaced it in the lateral direction and as a rule caused an impression in the cord. Only in three of the animals that had been given transvertebral injections (all in group 2) had the bulk of the paraffin remained on the ventral aspect of the cord and thereby caused an elevation of the latter corresponding approximately to one half to one normal spinal cord diameter. No dorso-ventral compression of the cord was demonstrable in these animals (fig 8). In all three animals subjected to laminectomy at T12 and L2 and injected with



Fig 11 Cross-section close to the disk T13/L1. The conditions of the experiment were identical to those that have been described under fig 9. After the recovery from anaesthesia the animal had signs of a moderate paresis (grade II) of both hind legs. The condition has since gradually improved and at the end of the observation time there was only a slight paresis (grade I). The animal was destroyed 4 weeks after the operation.

Ample formation of bone and cartilage on the inside of the remaining parts of the articular processes (observe especially the left of the picture). The remaining defect in the roof is filled with fibrous connective tissue. The cord and its meninges are elevated by a ventral paraffin mass to such an extent that the distance between the ventral surface of the dura and the bottom of the canal corresponds to approx.  $\frac{1}{4}$  of a normal cord diameter. In the vertical direction the space available for the cord is sufficient both in the median plane and to the right of it. To the left there is a dorso-lateral flattening of the cord corresponding to the proliferation of bone and cartilage from the inside of the articular processes.

paraffin via the laminectomy opening the paraffin mass lay ventral to the cord and elevated the latter approximately one half to one normal cord diameter. Neither in this group was there any considerable dorso-ventral cord compression; the cross-section of the elevated part of the cord showed however dorso-lateral flattening which was invariably most pronounced above the intervertebral disks. The shape of the cross-section of the cord was in good agreement with the borderlines of the newformed bone and cartilage on the inner surfaces of the remnants of the articular processes (figs 9, 10 and 11).

## DISCUSSION

The patho-anatomic findings are in good agreement with the assumption made in Part I that the dorso ventral flattening of the cord observed after excision of the dorsal portion of the arch (including the articular processes) is caused by shrinkage of granulation tissue formed during healing of the post laminectomy defect in the dorsal wall of the spinal canal. On scar contracture this tissue may form a membrane that is tightly stretched between its points of attachment to the cut surfaces of the arch thus compressing the cord ventralward. The above assumption is supported—*inter alia*—by the observation reported above that (after laminectomy with the A technique) the tendency towards spinal cord compression was considerably less in the thoracic region than at the thoraco lumbar junction and that this difference seems to be related to the fact that after laminectomy in the former region the tissue bridging the surgical defect was attached largely to the transverse processes and to the ribs i.e., to points situated at a high level in relation to the base of the spinal canal.

The main purpose of the current experiments was to elucidate the effect of healing on the spinal cord after extensive laminectomy with the two operative methods described in Part I when the cord had been displaced dorsally at the beginning of the healing process as it may be for instance by median herniation of a disk. In the first experimental series paraffin injections were given for technical reasons at the level of the disk between T10 and T11 instead of between T13 and L1 (The latter site of injection would have afforded a possibility of direct comparison with those experiments (Part I) in which laminectomy had been performed without paraffin injection). The thoracic laminectomy experiments were found to be unsuitable for elucidation of the above mentioned problem mainly for two reasons. On the one hand the tendency towards post laminectomy cord compression proved on the whole to be considerably less pronounced in this region than in the former operative region on the other hand at transvertebral injection the paraffin mass passed largely to the side of the median plane and thus did not bring about the desired elevation of the spinal cord. In cases where the cord actually was elevated moderate dorso ventral compression was demonstrable at patho-anatomic examination in those animals in which the dorsal and dorso-lateral parts of the arch had been completely excised with the A technique but in those where the outer parts of the arch had been spared at laminectomy with the B technique the cord deformation was limited to the aforementioned slight lateral or dorso-lateral flattening. The difference in the two operative methods with respect to the tendency towards compression *in this region* was not great enough to be reflected in a conspicuous difference in the functional behaviour of the spinal

cord during healing. In the supplementary experimental series the operation was performed in the region of T12 to L2 whereby, for technical reasons, the paraffin was introduced instead via the laminectomy opening and caused a substantial elevation of the spinal cord in all animals. In those of the last mentioned series of experiments which involved total excision of the dorsal and dorso-lateral portion of the arch the animals initially showed progressive improvement of the motor loss symptoms, but after approximately two weeks signs of rapidly progressing damage to the spinal cord appeared, and, after a further two weeks subtotal or total paralysis of the hind legs developed. Apart from symptoms of motor loss following upon the operation and progressively decreasing corresponding animals subjected to laminectomy with *retention of the outer compact bone of the dorso lateral part of the arch* showed no neurological symptoms over a similar period. In dogs of the first-mentioned group the spinal cord was found, at patho-anatomic examination to be compressed to a thin plate between the paraffin mass and the scar tissue, whereas the animals of the latter group showed no dorso-ventral flattening of the cord, even though the dorsal displacement was so great that the cord was situated largely in the excavated part of the arch. In these latter dogs moderate lateral or dorso lateral flattening of the cord was demonstrable. The last mentioned type of deformation seems to be caused for the main part by formation of bone and/or cartilage originating from the injured articular processes.

The results obtained seem to warrant the conclusion that the process of healing in dogs after extensive laminectomy with complete excision of the dorsal and dorso lateral parts of the arches including the articular processes (technique A) may entail a risk of severe spinal cord compression while this complication could be avoided when the dorsal portion of the arch including the articular processes had been spared at laminectomy (technique B). Whether it is possible with technique B to attain a *permanent* safeguard against secondary spinal cord compression—caused by scar shrinkage or proliferative processes—cannot be deduced from our results owing to the short observation times. Experiments with longer periods of observation in animals with spontaneous compression of the cord (herniated disks)—which experiments will be reported separately—suggest however that the risk of complications after type B laminectomy is inappreciable even in the long run. Should the abovementioned results be confirmed by further experiments with this operative method, it would thus be justifiable to restrict operations for median hernia to decompressive laminectomy and to retain the bulk of the prolapsed disks provided the operative technique is such as to afford an adequate safeguard against secondary dorsal spinal cord compression.

It was found that the abovementioned experiments in which transvertebral

injection of paraffin had been given in conjunction with thoracic laminectomy, did not permit comparison of the two surgical methods in respect to the risk of secondary compression of the spinal cord. They may, nevertheless be of some surgical interest for the following reason. Vaughan (1958) and Olsson (1960) hold the view that the surgical trauma at laminectomy performed on dogs when the spinal cord is already damaged may involve a risk of transforming a reversible into an irreversible lesion of the cord. The abovementioned experiments in which laminectomy was performed at a second operation *after the paraffin injection* suggest that the surgical trauma associated with this technique does not have any significant deleterious effect on the cord. The operations in question either had no effect whatsoever on the neurological status or caused only a transient impairment of motor function. In this connection however it must be pointed out that the operations were performed a relatively long time (8 to 17 days) after the initial trauma hence the experimental conditions were not altogether comparable to those obtaining in spontaneous spinal cord compression.

The experimental elevation of the cord by means of paraffin injection in the ventral epidural space *after opening the dorsal wall of the spinal canal at laminectomy*, may throw some light on the therapeutic significance of rhizotomy or division of the dentate ligaments in cases with corresponding degrees of spontaneous elevation of the cord by pathological processes. It has been considered that since the spinal nerve roots and the dentate ligaments fix respectively the dura to the base of the spinal canal and the spinal cord to the dura they may contribute substantially to the occurrence of spinal cord lesions associated with ventral expansive processes (Kahn 1947, Logue 1952, and Tovi & Strang 1960 among others). The fact that the motor disturbances in the experiments reported in B 3 were relatively slight and progressively abated throughout the observation period even though very pronounced elevation of the cord was present and no rhizotomy or division of dentate ligaments had been done suggests that the ventral fixation of the cord by the nerve roots and the dentate ligaments is not in dogs of major causal significance in spinal cord lesions caused by ventral compression at the junction of the thoracic and lumbar regions.

## SUMMARY

Laminectomy at the junction of the thoracic and lumbar regions (T12 to L2) was performed on dogs by a technique involving excision of the dorsal and dorso-lateral portions of the arch including the articular processes. In connection with laminectomy the spinal cord was elevated by means of an injection of paraffin wax into the ventral epidural space. During healing these animals

exhibited severe dorso-ventral compression of the spinal cord with total or sub-total paralysis of the hind legs. When the outer compact bone of the dorso-lateral portion of the arch including the main part of the articular processes was spared at laminectomy—the other experimental conditions being unchanged—dorso-ventral cord compression was prevented. At corresponding operations in the thoracic region (T9 to T12) the tendency towards cord compression was conspicuously less even after laminectomy with the first mentioned technique.

## REFERENCES

- Hahn E* The role of the dentate ligaments in spinal cord compression and the syndrome of lateral sclerosis. *J Neurosurg* 4 (1947) 191
- Logue V* Thoracic intervertebral disc prolapse with spinal cord compression. *J Neurol Neurosurg Psychiat* 15 (1952) 227
- Olsson S E* Diskbräck hos hund. *Medlemsblad f Sv Vet forbund* 12 (1960) 149
- Olsson S E* Ibid. 12 (1960) 185
- Toré D & Strang R R* Thoracic intervertebral disk protrusions. *Acta chir scand* 1960 suppl. 267
- Vaughan L C* Studies on intervertebral disc protrusion in the dog (4). *Brit vet. J* 114 (1958) 458















ACTA ORTHOPAEDICA SCANDINAVICA  
SUPPLEMENTUM No 57

---

*FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI CHIEF PROFESSOR K. E. KALLIO*

SURGERY OF ACUTE ARTERIAL INJURIES  
IN CONNECTION WITH FRACTURES  
AND DISLOCATIONS

*ERKKI V. S. KOSKINEN*

---

EJNAR MUNKSGAARD  
COPENHAGEN 1962





ACTA ORTHOPAEDICA SCANDINAVICA  
SUPPLEMENTUM No 57

---

FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI CHIEF PROFESSOR K. E. KALLIO

SURGERY OF ACUTE ARTERIAL INJURIES  
IN CONNECTION WITH FRACTURES  
AND DISLOCATIONS

ERKKI V. S. KOSKINEN

---

EJNAR MUNKSGAARD  
COPENHAGEN 1962

PRINTED IN FINLAND BY  
MERCATORIN KIRJAPAINO  
HELSINKI 1962

## CONTENTS

- I Introduction
- II Material with Case Reports
- III Arterial Trauma
- IV Skeletal Trauma
- V Treatment
- VI Discussion
- VII Summary and Conclusions
- VIII Zusammenfassung and Schlussfolgerungen
- IX Acknowledgements
- X References



## I INTRODUCTION

Acute traumatic arterial injury in connection with fracture is one of the most serious conditions as regards traumas of the extremities. As compared to other states causing insufficiency of circulation in the extremities such as aneurysm, arteriovenous fistulae or arteriosclerosis which are chronic of character and in which there are collateral paths, injuries of the blood vessels produced by trauma constitute an immediate menace to the extremity; they may result in loss of the extremity or in severe functional disturbance.

In comparison with the progress that has taken place in the field of traumatic and specially reconstructive surgery, arterial lesions in association with fractures have not met with the understanding and attention actually deserved by this severe complication. As a consequence many cases of this kind with fracture and arrest or disturbance of blood flow owing to arterial lesion have led to loss of the extremity or to disability on account of claudication, muscular atrophy, pareses and coldness. Not much interest has been attached to surgery of these injuries. The modern reconstructive surgery in this particular field has, however, created possibilities implying a decisive change of prognosis.

According to the material of *de Bakey* and *Simeone* (1) from World War II, simple ligation of the common iliac artery results in an incidence of 73 per cent amputation of the leg, ligation of the common femoral artery in 81 and of the superficial femoral artery in 54, and ligation of the popliteal artery results in 72 per cent gangrene and loss of the extremity. On the other hand, in series of arterial injuries from the time of the Korean war with treatment consisting of primary repair and reconstruction, loss of extremity ensued approximately in 15 per cent (5, 6) and in cases with major vascular lesion amputation became necessary later in 88 per cent only (7). These figures already serve to show that amputation of a considerable number of extremities could be avoided by applying reconstructive arterial surgery instead of ligation. In addition, later functional disturbances in the extremity, muscular atrophy, ischaemic contractures and paraesthesias occur in fewer instances (5).

Accidents occurring in civilian life provide more favourable conditions for repair of the injured extremity and it is appropriate to assume that the less destructive nature of most civilian injuries and particularly the shorter time within which definitive treatment can be instituted may even further improve the results.

Published reports on arterial injuries occurring in connection with fractures are rather few. Earlier reports dealt mainly with gangrene and amputation was the sole measure of treatment. Recently some successfully treated cases have been described in which the extremity could be saved (2, 10, 13, 14, 15, 17).

With respect to therapy we are confronted with a new problem because two therapeutic principles are concerned, namely those of vascular surgery and of bone surgery. This report concerns an effort to apply these principles to cases of traumatic arterial injuries associated with fractures and dislocations which would not be considered amenable to surgery by older standards. It is intended to attract greater attention to arterial injury in association with fractures and dislocations by pointing out the importance of this complication. An attempt to a more active therapeutic approach in view of restoration of blood flow in the injured extremity is essential. Thus it would be possible to create more favourable conditions for union of the fracture to take place.

## II MATERIAL WITH CASE REPORTS

The most essential aspects of the cases included in the present material relating to the type of the arterial injury and fracture to its preoperative symptoms and surgical treatment and to the results can be seen in Table 1

The material consists of 18 patients treated from 1960 to the beginning of 1962 in the Clinic for Orthopaedics and Traumatology University of Helsinki all but two of them (cases No 6 and 7) having been treated by the present writer In all instances the immediate arterial injury was an association of fracture or dislocation The injury of the cases has been generally of severe degree and in most cases the injury has created a critical condition as regards saving of the extremity None of the transient or slight peripheral ischaemic states have been taken into account During the time in question altogether 410 closed and open fractures of equivalent location have been treated in this hospital which received a great number of severe traumatologic cases This implies that severe arterial injury has accompanied skeletal trauma in 4.4 per cent of the cases In addition to arterial injury and skeletal trauma most patients were seriously traumatized in a state of shock and they had cerebral thoracic and abdominal traumata indicating surgical treatment Sixteen of the patients had not previously experienced any circulatory disturbances The history of two cases (No 1 and 13) contains slight symptoms of arteriosclerosis obliterans these patients however being able to do their daily work All patients were male most of them young in age The ages varied between 14 and 69 years

### Case Reports

The first four cases all of them due to traffic accidents displayed trauma of the femoral artery accompanying fracture of the femur In cases No 1 and 4 there was traumatic thrombosis in connection with the femur fracture

---

A brief report of this work has been presented at the meetings of the Finnish Surgeons Association on May 5 1962 and of the Finnish Orthopaedic Association on November 23 1961

TABLE 1 FRANCHIAL DATA ON THE MATERIAL OF THE PRESENT WORK

| No.                                    | Type of arterial injury | Site of fracture or dislocation | Associated injuries  | Recovery due to vascular signs and arterial graphic findings   | Surgical treatment   | Results   |
|--|-------------------------|---------------------------------|--|--|--|---|
| <b>Injured artery Femoral artery</b>   |                         |                                 |  |  |  |   |
| No. 60                                 | Thrombosis              | Compound femur fracture         | Cerebral contusion<br>Rupture of the kidney                        | Total pulses not palpable below the knee Total occlusion of the femoral artery Local haemorrhage developed   | Thromboendarterectomy Intramedullary nailing, talar transplantation            | Strong, dorsal pedal and posterior tibial artery pulses Motor and sensory function restored Gangrene healed Free passage in femoral and distal arteries Fracture consolidated |
| No. 16                                 | Obliteration            | Comminuted femur fracture       | Cerebral contusion   | Ischaemic paralysis of the leg with ulcerations coolness and claudication Dorsal pedal artery pulseless posterior tibial artery almost pulseless Obstruction of femoral artery | Healed with autogenous vein graft  | Strong peripheral pulsations warm extremity ulcerations healed in restricted graft Lower extremity fully completely including the distal artery Fracture consolidated         |
| No. 20                                 | Traumatic spasm         | Compound femur fracture         | Cerebral contusion<br>Intercranial haemorrhage                     | Extremity cold distally of the knee pulseless femoral artery angulated spastic at the point of fracture fills poorly   | Fluorination and advancement stripping, retrograde flushing, internal fixation | Dorsal pedal and posterior tibial arteries distinctly palpable Extremity warm Patient expired later as a result of cerebral haemorrhage                                       |
| No. 14                                 | Thrombosis              | Comminuted femur fracture       | Cerebral contusion<br>Traumatic haemorrhage                        | Cerebral pulsations not palpable Early deep gangrene of foot and lower leg Occlusion of femoral artery at the point of fracture  | Thoracotomy Skeletal traction Amputation                                       | Recovery Stump healed   |
| <b>Injured artery Popliteal artery</b> |                         |                                 |  |  |  |   |
| No. 62                                 | Rupture                 | Dislocation of knee joint       | Dislocation of femur<br>Acetabulum fracture<br>Fracture of the leg | Extremity cold distally of the knee pulseless Ischaemic paralysis Popliteal artery only fills with contrast medium up to the boundary of the femoral artery                    | Healed with Teflon prosthesis Retrograde flushing Amputation                   | Teflon prosthesis worked initially but progressive ischaemia resulted in gangrene Stump healed  |



|   |                 |                                |  |   |  |   |
|---|-----------------|--------------------------------|--|---|--|---|
| No 20   | Thrombosis      | Dislocation of knee joint      | None   | Ischaemic paralysis and early peripheral gangrene Occlusion of popliteal artery   | Thrombectomy<br>Flushing<br>Amputation   | Irreversible ischaemic state and gangrene Stump healed  |
| No 31   | Rupture         | Dislocation of knee joint      | None   | Far progressed gangrene of foot and lower leg Arteriography showed the popliteal artery to be severed at the knee joint gap   | Amputation   | Stump healed  |
| <b>Injured artery Anterior and/or posterior tibial arteries</b> |                 |                                |  |   |  |   |
| No 19   | Thrombosis      | Comminuted fracture of the leg | Cerebral contusion<br>Fracture of pelvis<br>Fracture of tibial condyle       | Foot cold and an aethetic disturbed motility Pedal pulses not palpable Arteriography showed occlusion of anterior tibial artery and spastic posterior tibial artery | Thrombectomy<br>Retrograde flushing<br>Internal fixation<br>Ilino bone grafting            | Strong pedal pulses Sensibility and motility intact Arteriography shows all arteries of the extremity filling without defect Ilino consolidation of the fracture  |
| No 18   | Thrombosis      | Compound fracture of the leg   | Cerebral contusion<br>Rupture of the liver<br>Fracture of humerus            | Peripheral ischaemic paralysis Pedal pulses not palpable Arteriographically occlusion of anterior tibial artery at the point of fracture                            | Thrombectomy<br>Retrograde flushing<br>Laparotomy Suture<br>Flon of liver rupture          | Cripher il pulses strong Foot warm Sensibility and motility normal Arteriography shows all arteries of the extremity filling without defect Fracture consolidated |
| No 10   | Traumatic spasm | Compound fracture of the leg   | None   | Foot cold and anaesthetic restricted motility Peripheral con fracture Anterior tibial artery pulseless dor al pedal artery almost pulseless                         | Liberation and adven<br>titia stripping Inter<br>nal fixation with fibula<br>transposition | Foot warm sensibility restored is well is motility Slight residu il con fracture of toes Strong pulsations Fracture consolidated                                  |
| No 23   | Traumatic spasm | Compound fracture of the leg   | Fracture of the leg  | Pedal pulses not palpable Arteriographically all arteries of the leg, angulated spastic at the point of fracture not filling in the region of the foot              | Asclotomy<br>Cutaneous trans<br>plantation   | Foot vital and warm Gangrene on the medial side healed Fracture consolidated  |
| No 16   | Thrombosis      | Compound fracture of the leg   | Cerebral contusion<br>Intracranial haemorrhage<br>Compound fracture of femur | Pedal pulses not palpable Extremity cold pale distally of the middle of lower leg Arteriographically the arteries fill only to the point of fracture                | Thrombectomy<br>Retrograde flushing<br>Craniotomy of sub<br>dural haematoma                | Peripheral pulsations palpable ex tremity warm Patient expired as a result of cerebral lesion   |

| Case No.                       | Type of arterial injury | Bone fracture or dislocation            | Vasculature of injury | Irradiation of arterial supply  | Surgical treatment   | Results  |
|--------------------------------|-------------------------|---|-----------------------|---|--|--|
| No 14                          | Thrombosis              | Compound fracture of the leg            | None                  | Early peripheral gangrene. Cold pulses not palpable. Arterio-venous anastomosis in femoral artery and complete arrest of blood flow at the point of the fracture  | Amputation   | Stump healed   |
| No 15                          | Traumatic               | Compound fracture of the leg            | Fracture of the leg   | Imminent peripheral gangrene. Cold arteries palpable. Arterio-venous anastomosis in femoral artery and complete arrest of blood flow at the point of the fracture | Fluorination and active drainage, intramedullary nailing         | Peripheral pulses strong. Sensibility and mobility normal. Fracture consolidated                       |
| No 16                          | Traumatic               | Compound fracture of the leg            | None                  | Peripheral ischaemic condition. Arterio-venous anastomosis in femoral artery and complete arrest of blood flow at the point of the fracture                       | Ligamentary sympathectomy. Internal fixation and bone graft      | Cold pulses palpable. Fracture consolidated  |
| No 17                          | Traumatic               | Dislocation of ankle                    | None                  | Partial pedal artery. Pulses in tibial posterior artery almost palpable. Cold cold pulse  | Decompression. Open reduction                                    | Strong, pulsations. Cold warm good function  |
| Injured artery Brachial artery |                         |   |                       |   |  |  |
| No 18                          | Rupture                 | Fracture dislocation of the elbow joint | None                  | Peripheral ischaemic paralysis. Radial and ulnar arteries pulseless   | Resection and arterial anastomosis. Resection of caputulum radii | Radial artery pulse strong. Arterio-venous anastomosis in brachial ulnar and radial arteries           |
| No 19                          | Thrombosis              | Compound fracture of humerus            | None                  | Extremity cold. Pulses in artery finger gangrene. Incomplete filling of brachial artery at the point of fracture  | Fluorination. Arterio-venous anastomosis. Bone transplantation   | Extremity warm. Pulsation of radial artery palpable. Inner stump healed. Consolidation of the fracture |

in the first case it was treated by thrombectomy and thromboendarterectomy and medullary nailing of the fracture was performed at the same time. In case No 4 restoration of circulation was contraindicated on account of other injuries and the extremity was lost. In case No 2 the obstruction ensuing after rupture of the femoral artery was corrected by autogenous vein graft and in case No 3 circulatory disturbance produced by traumatic spasm of the artery was treated by liberation and adventitia stripping.

*Case No 1* F.C. farmer aged 69. As a result of a traffic accident there was on the right side a compound dislocated fracture of the femur and injury of femoral



Fig 1 Case No 1 Traumatic thrombosis of the femoral artery in association with compound femur fracture causing peripheral ischaemic paresis and gangrene

Fig 2 Case No 1 Postoperative arteriogram three months after thrombectomy and thromboendarterectomy of the femoral artery. Internal fixation of the fracture by medullary nailing was done in connection with arterial surgery



Fig. 3 Case No. 1 Gangrene as a result of traumatic thrombosis of the femoral artery in association with femur fracture

Fig. 4 Case No. 1 Ten months after arterial surgery and medullary nailing the ischaemic state has been corrected arterial pulsations in the extremity are strong. The fracture is consolidated scars are visible

artery cerebral contusion and rupture of the kidney. Foot and lower leg were cold and there was no demonstrable sensory or motor function below the knee. Popliteal and pedal pulses could not be palpated and arteriography revealed an obstruction in the femoral artery at the point of fracture (Fig. 1). The patient's condition was critical owing to associated traumata and not until two weeks after the accident when there were deep gangrenes (Fig. 3) in calcaneus and lower leg was thrombectomy of femoral artery performed as well as intramedullary nailing of the femur. — At the point of fracture the femoral artery was hard and pulseless in conformity with the arteriographic findings. The arteriotomy wound was filled by a fresh thrombus which continued proximally and distally as a thickening of the intima. Upon removal of the thrombus 15 cm in length and upon thromboendarterectomy blood flow was completely free. The leg was warm after the operation and pedal pulses could be

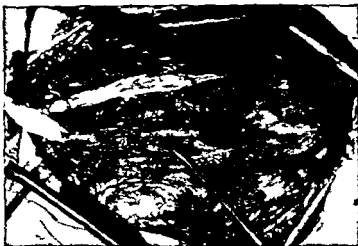


Fig 5 Case No 1 Obstruction of the femoral artery after rupture and ligation in connection with femur fracture. Severe ischaemic state, ulcerations, coldness and claudication ensued upon the injury. — At operation three months later the femoral artery was strongly adherent to the bone, angulated, pulseless and occluded.



Fig 6 Case No 2 Restoration of blood flow was achieved by autogenous vein transplantation.



Fig. 3 Case No. 1 Gangrene as a result of traumatic thrombosis of the femoral artery in association with femur fracture



Fig. 4 Case No. 1 Ten months after arterial surgery and medullary nailing, the ischaemic state has been corrected, arterial pulsations in the extremity are strong. The fracture is consolidated, scars are visible

artery, cerebral contusion, and rupture of the kidney. Foot and lower leg were cold and there was no demonstrable sensory or motor function below the knee. Optimal and pedal pulses could not be palpated, and arteriography revealed an obstruction in the femoral artery at the point of fracture (Fig. 1). The patient's condition was critical owing to associated traumata and not until two weeks after the accident, when there were deep gangrenes (Fig. 3) in calcaneus and lower leg, was thrombectomy of femoral artery performed, as well as intramedullary nailing of the femur. — At the point of fracture the femoral artery was hard and pulseless, in conformity with the arteriographic findings. The arteriotomy wound was filled by a fresh thrombus which continued proximally and distally as a thickening of the intima. Upon removal of the thrombus, 15 cm in length, and upon thromboendarterectomy, blood flow was completely free. The leg was warm after the operation and pedal pulses could be

strongly felt. One week later excision of necrosis and cutaneous transplantation was made. Arteriography three months later (Fig 2) showed that the femoral and distal arteries filled rapidly and well. Ten months after the operation (Fig 4) there were no signs of circulatory insufficiency in the extremity; the fracture had consolidated and the patient had returned to his normal mode of life.

*Case No 2 S J painter aged 16.* As a result of traffic accident the patient had a fracture of the left femur and cerebral contusion. The leg was cold, the foot insensible and could not be moved. In connection with nailing of the femur in the local hospital it was mandatory to ligate the femoral artery owing to rupture and profuse haemorrhage. On account of insufficiency of arterial circulation the patient was transferred to our clinic after three months when ischaemic paralysis of the leg, peripheral ulcerations and strong muscular atrophy and claudication were ob-



Fig 7 Case No 9 Arteriogram three months after the autogenous vein grafting in obstruction of the femoral artery showing good function of the artery.

Fig 8 Case No 2 Nine months after the arterial surgery. There are no symptoms of arterial insufficiency; peripheral pulsations in the extremity are strong and the fracture is consolidated.

served. Arteriography showed a constriction in the artery below the middle of the femur at the point of fracture and deficient filling in the peripheral arteries. Operation revealed that in the region indicated by the arteriography the femoral artery was strongly adherent to the underlying femur at the very point of fracture which had not consolidated. The artery was narrow, hard, pulseless on a length of 10 cm (Fig. 5). Proximally and distally of this section it had soft consistency and its pulsation could be felt. Arteriotomy revealed thick deposits in the intima; after their removal there was only minimal blood flow. Autogenous vein graft was performed using the vena saphena end to side (Fig. 6). The graft worked well and the leg was warm after the operation; strong pedal pulses were palpable. Contrast arteriography three months later (Fig. 7) showed good and rapid filling in the entire femoral artery continuum into the distal arteries. The necrotic wounds



Fig. 9 Case No. 3 Traumatic vasospasm of the femoral artery in association with open fissure femur fracture. The circulation of the extremity is seriously deranged as a result of the arterial injury.

Fig. 10 Case No. 4 Arteriogram revealing a defect in the femoral artery at the point of fracture in association with femur fracture.



on the lower leg and in the metatarsal region healed and closed rapidly. On follow up examination after nine months the fracture was clinically consolidated and the patient had no symptoms of arterial insufficiency in his leg. Dorsal flexure of the foot was slightly reduced but the patient was able to move freely and to work (Fig. 8).

*Case No. 3* L.R. welder aged 26. As a result of traffic accident the patient had a severe compound fracture of the right femur and a cerebral lesion. Two hours later when the patient was brought into the hospital he was under shock. The right thigh was markedly swollen, deformed with a gaping wound on the inner side. The leg was cold, pulseless and could not be moved by the patient. X-rays revealed dislocated fracture of the femur which displayed a shortening. On ateriography (Fig. 9) the femoral artery was found to bend medially at the point of fracture and to be narrowly filling and pastic over about 15 cm. On exploration carried out after the general condition had improved the femoral artery was very narrow and no pulsation could be observed. After performed liberation, adventitia stripping and treatment with warm saline the artery became wider and pulsation was obtained. There was no thrombosis. At the same time internal fixation of the fracture was carried out. After the operation the leg was warm and the peripheral pulsations were clearly palpable. However the patient expired later as a result of cerebral haemorrhage.

*Case No. 4* J.Y. machinist trainee's son aged 14. As a result of traffic accident the patient had a fracture of the right femur and traumatic thrombosis of the femoral

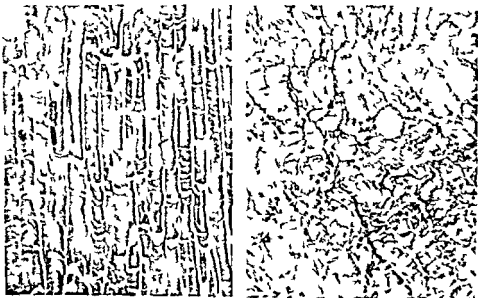


Fig. 11 Case No. 4 Typical muscular necrosis as a result of the ischaemic state. Muscle fibres can be recognized in the photomicrograph but there are no nuclei (Left). — On the right the micrograph of a biopsy from the boundary between healthy and necrotic tissue  $\times 120$ .



Fig. 1. Case No. 4 Photomicrograph of the femoral artery revealing proximally to the thrombosis strongly thickened intima and new formation of connective tissue. This is a result of trauma to the arterial wall.  $\times 60$

artery. Moreover cerebral injury and thorax compression displacement of mediastinum causing breathing difficulties and indicating thoracostomy. Operation of the artery was counterindicated by these injuries. A line of demarcation developed in the region of the ankle and deep necrosis in the lower leg. Arteriography (Fig. 10) revealed a contrast medium defect at the point of fracture in the femoral region. Pyletic and posterior tibial arteries filled well but the anterior tibial artery was not filled in its distal part. Syme's amputation was performed after three weeks but on account of poor healing of the stump amputation of the lower third of the thigh had to be done after two months. Examination of the leg showed that the greater part of all its muscles were in necrosis (Fig. 11) even above the knee as far as the quadriceps muscles. This finding was surprising as compared with the arteriography but it is explained by acute femoral artery obstruction (Fig. 12) with subsequent irreversible ischemia aggravated by a severe state of shock.

The next three cases are injuries of the popliteal artery. In case No. 5 anterior dislocation of the knee joint resulted in severance of the artery. In case No. 7 the same result was produced by compression of the knee and lateral dislocation. The thrombosis in case No. 6 was a sequel of a major hyperextension injury. Reconstruction with a Jefferson prosthesis was done in the first mentioned case and thrombectomy in case No. 6. However



Fig 13 Case No.      Serious dislocation of the knee joint causing transection of the popliteal artery.

Fig. 14 Case No.      Arteriograph showing arrest of the contrast medium in the popliteal artery above the knee joint.

definitive treatment could only be instituted at a later stage and the prognosis was bad.

**Case No. 17 B** farm worker aged 62. The patient fell from a tractor and sustained anterior dislocation of the right knee joint (Fig 13) and rupture of the popliteal artery. Moreover luxation of femur acetabulum fracture and fracture of the left lower leg. Arteriography revealed filling with contrast medium up to the boundary between the popliteal and femoral arteries (Fig 14). Exploration showed the popliteal artery to be severed altogether. Although more than two days had passed since the accident an attempt at reconstruction was made with a Teflon prosthesis by the end to end technique. The distal thrombus was removed by retrograde flushing and good pulsation was achieved through the prosthesis which worked (Fig 15). On the next day the dorsal pedal pulse could be felt but there was still insufficiency of circulation. At repeated exploration the points of anastomosis were free but progressive peripheral ischaemia ensued all the same and supracondylar amputation was performed on the tenth day.



Fig. 15 Case No. 5 Teflon prosthesis of the popliteal artery at operation

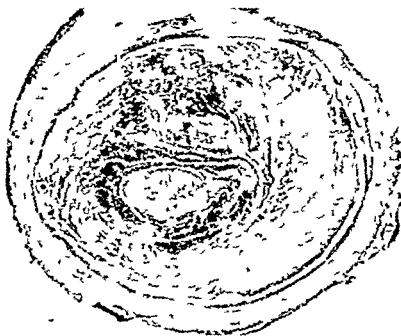


Fig. 16 Case No. 6 Photomicrograph showing thrombosis of the popliteal artery resulting from serious dislocation of the knee joint (60)

Case No. 6 A M telephar lineman, aged 30. While he was up a pole the pole fell and the patient's left knee was forced into hyperextension. In the local hospital dislocation of the knee joint was established. Injury of the popliteal artery was suspected and the patient was sent to our hospital. Now on the tenth day after the

injury arteriography revealed an obstruction in the popliteal artery 5 cm proximally of the joint gap. On exploration the artery was hard pulseless. The intima was thickened, a thrombus 8 cm in length was removed and the distal branches were flushed. As could be expected no improvement of circulation ensued, gangrene developed and the lower part of the thigh was amputated (Fig. 16).

*Case No. 7* M. K. lorry driver aged 31. The patient's left leg was caught between the lorry and trailer resulting in open dislocation of the knee, profuse haemorrhage and shock, of which the patient recovered in the local hospital. Arteriography showed the popliteal artery to be severed at the knee joint gap; in the lower leg no filling was to be seen excepting the occurrence of fine collaterals down to about its middle. The foot became necrotic and amputation of the lower third of the thigh was done one week after the accident. Exploration of the amputated leg showed that the popliteal artery had been totally severed just proximally of its branching point.

In nine cases (No. 8 to 16) there was a traumatic injury of the anterior tibial and/or posterior tibial artery accompanying fracture of the lower leg. All but one were caused by traffic accidents. In cases No. 8 and 9 there was a thrombus of the anterior tibial artery, and in case No. 12 thrombosis of the anterior and posterior tibial arteries. In all of the cases circulation could be restored and the leg saved by thrombectomy and retrograde flush manoeuvre. In cases No. 10 and 14 the circulatory disturbance produced by compression of anterior and posterior tibial arteries and by traumatic spasm was treated by liberation of the arteries and adventitia stripping. A case (No. 15) with traumatic spasm of the distal tibial arteries was successfully treated by high lumbar sympathectomy. In case No. 11 the ischaemic condition of the extremity was relieved by fasciotomy. In case No. 13 there was a thrombus of the anterior and posterior tibial arteries and the leg was lost. In this case the traumatic arterial injury occurred in a leg with previous arteriosclerotic changes of the femoral artery. In one case (No. 16) injury of the anterior tibial artery was caused by severe dislocation of the ankle.

*Case No. 9* S.S. type setter aged 19. In a traffic accident the patient was hurled 10 m by a car. Comminuted, dislocated fracture of the right lower leg, fracture of the condyle of tibia, fracture of the pelvis and cerebral contusion were established. The foot was cold and anaesthetic. Pedal pulses could not be palpated. Arteriography revealed that the anterior tibial artery did not fill distally of the fracture and the posterior tibial artery was spastic. At exploration the artery was pulseless. Arteriotomy was performed, removing a fresh thrombus and retrograde flushing was done. At the same time internal fixation of the fracture and iliac bone graft were carried out. — The artery pulsated well after the operation. Contrast arteriography 22 months later showed that femoral, popliteal and anterior and posterior tibial arteries filled well without gaps in the entire region (Fig. 17). The fracture had consolidated and the leg functioned well.



Fig. 17 Case No. 8 Example of a case with high traumatic thrombosis of anterior tibial artery and spasm of posterior tibial artery in association with fracture of the lower leg and consequent imminent peripheral gangrene. Thrombectomy and retrograde flush out manoeuvre were performed with simultaneous internal fixation of the fracture and iliac bone graft. — The arteriogram has been made 22 months after the operation. Anterior and posterior tibial arteries are strongly palpable, bony consolidation of the fracture has taken place and function of the leg is good.

**Case No. 9.** A R. handyman, aged 18. As a result of a traffic accident, the patient sustained a compound fracture in the upper part of the right lower leg, fracture of the upper arm, rupture of the liver and cerebral contusion. Clinically, imminent distal gangrene of the extremity could be observed and arteriography revealed the anterior tibial artery to be broken at the level of the fracture. Posterior tibial and fibular arteries were thin and filled poorly. Upon appropriate treatment for shock, arteriotomy and thrombectomy of the anterior tibial artery was performed and good blood flow was obtained. The fracture was repositioned and placed in a well padded plaster cast. After this manipulation, pedal pulses were strongly palpable and the

extremity was warm. Furthermore laparotomy was performed and the rupture of the liver sutured. — Arteriography three months later showed completely free blood passages in the lower extremity. The fracture had consolidated and the function of the leg was good.

*Case No 10* S R tractor driver aged 19. The trouser leg of the patient's coveralls caught on the transmission shaft of the tractor resulting in compound fracture of the right lower leg and arterial lesion. On admission for treatment the foot was cold and partly anaesthetic; there was only minimal motility of ankle and toes. Posterior tibial artery was pulseless and dorsal pedal pulse weak. Exploration revealed the bone fragments to be dislocated so that the anterior and posterior tibial arteries were compressed and angulated. Hardly observable pulsation existed. The arteries were liberated and adventitia stripping was performed whereupon strong pulsation was obtained. The fragments were repositioned and internal fixation was carried out with a fibula graft. — Six months later the peripheral pulsations were strong. The fracture had consolidated and the function of the leg was good.

*Case No 11* P K electrical fitter aged 23. Sustained fracture of both tibiae in a traffic accident. On the left side with compound fracture imminent gangrene of the leg was at once observable. Foot and toes were anaesthetic and pale; no movements were possible. Arteriography revealed at the site of the fracture in the middle third angulation and dislocation of both tibial arteries and a break distally. The vascular condition did not improve on reduction. Fasciotomy was performed and the compressing haematoma was removed. Quite rapidly hereafter the ischaemic condition was relieved; sensation and motility were restored.

*Case No 12* M S draftsman aged 16. Sustained in a traffic accident compound fractures of the right femur and lower leg. The extremity was cold; dorsal pedal and posterior tibial arteries were pulseless. Cerebral symptoms indicated intracranial haemorrhage. The state of shock was treated after which a subdural haematoma was removed from the right hemisphere and the anterior and posterior tibial arteries of the right leg were exposed. They were pulseless and spastic. The haematoma compressing the arteries was evacuated and a thrombus removed by retrograde flush from both arteries after which pulsation returned and could be palpated after the operation. As a result of the cerebral lesion however the patient died after two days. — In the preoperative arteriography the femoral artery filled well past the point of the femur fracture; anterior and posterior tibial arteries were only filled to the level of the leg fracture and there was no arterial filling beyond this.

*Case No 13* A B worker aged 63. In a motorcycle accident the patient sustained a compound fracture of the left lower leg. There had previously been symptoms of claudication in his left lower extremity. Ten days later the leg was cold without peripheral pulsation. Arteriography revealed obstruction of femoral artery and complete arrest of blood flow at the fracture level. Gangrene of the leg developed gradually despite sympathetic blocks and heparin; this was a result of fracture of the initially poorly vascularized extremity (Fig 18). Femur amputation was performed 16 days after the accident. The amputated leg was examined and there was arterial and venous thrombosis proximal of the fracture. The condition *in situ* correlated with the arteriogram made prior to operation and with the arteriogram from the amputation specimen (Figs 19 and 20).



Fig. 1 - Case No. 3 Example of a case with high traumatic thrombosis of anterior tibial artery and spasm of posterior tibial artery in association with fracture of the lower leg, and consequent imminent peripheral gangrene. Thrombectomy and retrograde flush-out manoeuvre were performed with simultaneous internal fixation of the fracture and iliac bone graft. - The arteriogram has been made 22 months after the operation. Anterior and posterior tibial arteries are strongly palpable. bony consolidation of the fracture has taken place and function of the leg is good.

Case No. 9 A fit handyman aged 18. As a result of a traffic accident the patient sustained a compound fracture in the upper part of the right lower leg, fracture of the upper arm, rupture of the liver and cerebral contusion. Clinically imminent distal gangrene of the extremity could be observed and arteriography revealed the anterior tibial artery to be broken at the level of the fracture. Posterior tibial and fibular artery were thin and filled poorly. Upon appropriate treatment for shock, arteriotomy and thrombectomy of the anterior tibial artery was performed and good blood flow was obtained. The fracture was repositioned and placed in a well padded plaster cast. After this manipulation pedal pulses were strongly palpable and the



*Case No 14 A B* gardener aged 21 In a traffic accident sustained bilateral fracture of the leg On the right side with open fracture imminent gangrene was observable Peripheral pulsations could not be palpated In arteriography arrest of the anterior tibial artery at the level of the fracture posterior tibial artery was plastic and thin Both tibial arteries were immediately exposed they were dislocated by fragments pulseless and narrow After liberation adventitia stripping and heparin saline flushing the arteries dilated and pulsation reappeared The fracture was repositioned and intramedullary nailing was performed The fracture consolidated and good vascular function ensued

*Case No 15 A O* mechanic aged 24 In a traffic accident sustained fracture of the right leg Ischaemic condition developed rapidly the peripheral arteries were almost pulseless foot and leg were cold partly anaesthetic Arteriography showed the arteries of the leg to be angulated at the level of fracture distally from this point only posterior tibial artery poorly filled Early strong osteoporosis in bones of leg and talus Since the vascular lesion was fairly distal high lumbar sympathectomy was performed which considerably relieved the ischaemic condition One week later internal fixation of the fracture and iliac bone graft were performed The fracture consolidated rapidly and good function of the leg was obtained

*Case No 16 K T* worker aged 35 A fall from a high building resulted in serious dislocation of ankle and talus This caused compression and stretching of the anterior tibial artery and circulatory disturbance in the foot At operation the dislocation obstructing circulation was corrected whereupon the condition of the foot improved After six months the function of the foot was good and there were no signs of circulatory disturbance

Two cases of the present material (No 17 and 18) concern injury of the brachial artery caused by posterior fracture dislocation of the elbow joint in one case (No 17) and by compound fracture of the upper arm in the other (No 18)

*Case No 17 E V* tradesman aged 26 A fall from great height resulted in posterior fracture dislocation of the elbow joint The peripheral part was cold numb pulseless extensive haematoma at the elbow joint On immediately performed exposure the brachial artery was found to be completely severed the ends thrombosed Resection and arteriorrhaphy were performed after which the radial artery pulse was strong The fractured capitulum radii was removed and the dislocation corrected Later arteriography showed brachial ulnar and radial arteries filling well without arrest the joint had a good range of motion (Figs 21—24)

*Case No 18 J K* mechanic aged 33 Sustained in a traffic accident a compound fracture of the right upper arm with traumatic spasm of the brachial artery and thrombosis In spite of intensive vasodilatory treatment the fingers became necrotic owing to blocks of the stellate ganglion The entire extremity was cold with reduced sensibility The arteriogram revealed incomplete filling of the brachial artery distally of the fracture level On exploration two months later the artery was found to be surrounded by thick scar tissue and there was pulsation only halfway down the upper



Fig. 21 Case No. 1. Posterior fracture dislocation of the elbow joint which caused total rupture of brachial artery.

Fig. 22 Case No. 1. Arteriogram after resection of thrombosed ends of ruptured brachial artery and arteriorrhaphy. Fractured capitulum radii has been excised and dislocation corrected.



Fig. 23 Case No. 17. Rupture of brachial artery in connection with fracture dislocation of the elbow joint.

Fig. 24 Case No. 1. Restoration of blood flow achieved by excision of thrombosed ends of the artery and arteriorrhaphy.

arm to the point of fracture. The artery was exceedingly hard walled, narrow and spastic. After adventitia stripping and flushing pulsation reappeared in the entire artery. The changes in the vessel were of the kind encountered in an old thrombosis with recanalization. Control arteriography showed that the artery filled. Four months later the extremity was warm, but only fibrous consolidation of the fracture had formed. A bone traction was required, which resulted in osseous consolidation.

### III ARTERIAL TRAUMA

While most direct vascular lesions are associated with penetrating wounds and lacerations the principal types of acute arterial injury associated with skeletal trauma are 1) arterial contusion and subsequent traumatic vasospasm 2) thrombosis and 3) arterial rupture

The aetiology mechanism and surgical aspects of each type will be reviewed in the following paragraphs and related to the cases of the present material

#### Arterial Contusion and Traumatic Vasospasm

Traumatic vasospasm is a consequence of crush type injury caused by the fracture. A fragment or fracture haematoma may mechanically compress the blood vessel or the spasm may be the result of stretching of the artery. When the blood vessel is explored signs of contusion in the adventitia can be seen. It is reduced in size and adherences to the surrounding tissues may have formed. In acute arterial spasm examination in the intimal region reveals no changes.

In case No. 3 typical contusion induced traumatic vasospasm of femoral artery had ensued after a serious compound fracture of the right femur. At clinical examination two hours later the extremity was cold distally of the knee. popliteal and pedal pulses were not palpable. Arteriography showed the femoral artery to be angulated medially at the point of fracture, narrow and slowly filling (Fig. 9). On exploration the muscle tissues at the point of fracture were found to be lacerated, the artery displaced from its usual location and surrounded by an extensive haematoma. On closer examination the adventitia showed signs of contusion, there were no lacerations, but the artery was considerably constricted and pulseless at the point of fracture. After liberation, adventitia stripping and treatment with warm saline the artery was dilated and pulsation reappeared. Also the peripheral pulsation could be palpated after the operation and the extremity was warm.



Fig. 21 Case No. 1. Posterior fracture-dislocation of the elbow joint, which caused a total rupture of brachial artery.

Fig. 22 Case No. 1. Arteriogram after resection of thrombosed aneurysm of ruptured brachial artery and arteriohematoma. Fractured capitulum radii has been excised and dislocation corrected.



Fig. 23 Case No. 1. Rupture of brachial artery in connection with fracture-dislocation of the elbow joint.

Fig. 24 Case No. 1. Restoration of blood flow achieved by excision of thrombosed aneurysm of the artery and arteriohematoma.

arm to the point of fracture. The artery was exceedingly hard wall and narrow and spastic. After a venotomist stripping and flushing, pulsation reappeared in the entire artery. The changes in the vessel were of the kind encountered in an old thrombosis with recanalization. Control arteriography showed that the artery filled. Four months later the extremity was warm. But only fibrous consolidation of the fracture had occurred. A bone transplantation was required, which resulted in osseous consolidation

It is obvious that the retarded circulation caused by the thrombus at the point of lesion and the time needed to improve the patient's general condition before restoration of circulation contributed to the spread of the thrombosis. In this instance collateral circulation sufficed to maintain blood flow in the extremity although acute occlusion of the main artery was concerned.

Thrombosis is not rare either as a consequence of fracture of the lower leg. It is well known that the upper part of the tibia is a dangerous area for fracture to occur with respect to arterial injury owing to the proximity of the popliteal artery and of the bifurcation to the bones. *Watson Jones* (16) mentions that of seven high oblique leg fractures with displacement five were complicated by gangrene of foot and leg. In an earlier work the author (8) has reported cases of fracture of the lower leg with gangrene resulting from arterial injury. Of the eight fractures of the lower leg in the present material thrombosis caused severe acute disturbance of circulation in four. In all but one of them restoration of the blood flow could be achieved and the extremity was saved.

Case No. 8 is a typical case of fracture of the lower leg and associated arterial lesion in which imminent gangrene could be avoided by removing a thrombus from the anterior tibial artery and by retrograde flush. At the same time internal fixation of the fracture was carried out. At follow up examination two years later the dorsal pedal and posterior tibial pulses could be strongly felt, the fracture showed bony consolidation and the leg displayed good function. Arteriography revealed that all three arteries of the lower leg filled well with contrast medium (Fig. 17).

### Arterial rupture

Transsection or perforation of the artery is not as common in association with fracture as it is in open lesions of the blood vessels, e.g. in stab or shot wounds. Traumatic arterial rupture in connection with diaphyseal fracture is less common than thrombosis, but it may be a consequence of serious injury and dislocated fracture or fracture dislocation. A sharp fragment may produce rupture of a vessel and this may secondarily cause pulsating haematoma, traumatic aneurysm or arteriovenous fistulation.

Profuse haemorrhage may force arterial ligation to be employed as an emergency measure, as can be seen in case No. 2. In this illustrative case a serious traffic accident resulted in rupture of the femoral artery, which

had to be ligated in connection with the medullary nailing of the femur owing to haemorrhage. Primarily ischaemic paralytic was observable succeeded by severe peripheral disturbance of circulation coldness ulcerations and elimination symptoms. Moreover healing of the fracture was delayed and only a fibrotic callus was demonstrable at the time of the arterial reconstruction by means of autogenous vein graft. Three months later the artery was strongly adherent at the point of fracture impaled pulseless and very narrow (figs. 7 and 6). This case is an example of the vulnerability of the femoral artery in fracture of the distal part of the femur owing to their close anatomical relation. In fractures in this region and particularly if there is dislocation the possibility of arterial lesion should always be kept in mind and prepared for. Total rupture of the brachial artery in connection with fracture dislocation of the elbow joint is demonstrated by Case No. 17 (figs. 21—24). Restoration of blood flow and good function of the joint were achieved by immediately performed arterial surgery and bone surgery.

In connection with injuries dislocations and fractures of the knee region arterial ruptures surpass thrombosis in frequency owing to the peculiar anatomic conditions in the popliteal region. The popliteal artery passes distally between the heads of the gastrocnemius muscle and then beneath the tendinous arch of the soleus muscle by which it is anchored firmly against the underlying bone. At this point at the bifurcation the artery branches into the anterior and posterior tibial arteries the first of which passes immediately through the interosseous membrane. Any distortion of the skeletal anatomy causes stretching of the popliteal artery. This occurs particularly when the tibia is dislocated forward with respect to the femur or in serious hyperextension injuries. When the knee joint or a fracture is dislocated the communicating arteries poorly protected by soft tissues are also likely to be damaged. Distal circulation remains very unsatisfactory and the prognosis with respect to viability of the extremity is poor. In the material recently reported by Hoover (4) in which altogether 12 cases of popliteal arterial injury have been collected from the years 1912—1960 six of them presenting rupture gangrene and amputation ensued in eleven cases. In the one remaining case the surviving limb was subsequently cool and anaesthetic.

In the three cases of popliteal arterial injury of the present material complete transection of the artery was concerned in two and thrombosis in one. In all cases the trauma was severe in degree and definitive treatment could not be instituted until at a later stage so that the prognosis was bad. One of the complete transections of the artery was corrected by a Leflon

prosthesis and retrograde flush was applied. However, distal thrombosis was so far progressed that circulation could no longer be restored although the graft worked. In the other two cases the ischaemic state had already developed to the stage of gangrene in the peripheral parts and they terminated in amputation.

#### IV SKELETAL TRAUMA

In the present material all fractures with associated vascular lesion with the exception of one fracture of the humerus and one fracture-dislocation of the elbow joint were fractures or dislocations of the lower extremities. In the entire series of 18 cases the different sites of fracture or dislocation were represented by the following numbers of cases: femur — 1, knee joint

3, tibia or and fibula — 8, ankle — 1, humerus — 1, and elbow joint — 1.

Ten patients had a compound fracture while there was no wound in the leg connected with the fracture in the other eight. Generally considered the injury was serious in degree in all cases which is indicated except by the relatively high number of compound fractures above all by the fact that most patients had associated injuries and were in shock on admission. Most bone injuries were comminuted fractures and all presented dislocation on admission. The fragments were in most cases greatly displaced, the lesion in the soft parts was remarkable and there was shortening of the extremity. All femur fractures had their site in the distal part of the thigh while the location of the fracture in the lower leg varied so that it was in the upper third in two cases, in the middle third in two cases and on the boundary between the middle and lower thirds in another two cases. In contrast to earlier investigations in which the upper part (16) and the lower part (8) of the lower leg were found to be dangerous locations of fracture as regards arterial lesion, no distinct predisposition in this respect was evident in the present cases. In all dislocations of the knee joint there was rupture of the subcutaneous soft tissues surrounding the knee joint and the degree of dislocation was considerable. It appears obvious that the extent of dislocation and the consequent damage of soft tissues which otherwise have a protective effect play an important part in the occurrence of arterial injury.

Owing to anatomical relations in certain regions even less pronounced dislocation may produce arterial lesion e.g. in the distal part of the femur where the femoral artery lies close to the bone in Hunter's canal. The popliteal area and the supracondylar and elbow region of the upper arm are well known to be zones of fracture or dislocation involving great danger to arteries. The humerus fracture in the present material was located distally of the middle part of the bone.



## V TREATMENT

Combined arterial injury and bone trauma in an extremity implies combined treatment aiming simultaneously at restoration of blood flow and bony consolidation of the fracture in this order. Of course therapeutic measures dictated by immediate peril to the patient's life must take precedence over such treatment no matter what the consequences may be for the extremity.

For restoration of the blood flow methods are employed which enable circulation to be reestablished by surgical means and on the other hand measures conducive to maximum establishment of collateral circulation in order to secure blood supply to the extremity. All conservative measures of treatment have the last mentioned object and they include the use of anticoagulants, vasodilatory agents and sympathetic blocks. The extremity may retain its life depending on the location, kind and degree of severity of the arterial lesion, but when the main artery is eliminated from circulation at least relative ischaemia will remain which causes its typical symptoms of claudication, sensibility to cold and various motor and sensory disturbances. A segmental arterial obstruction of this kind which can be achieved as final result is equivalent to the state in which the patients come under treatment in chronic obliterative arteriosclerosis.

When there is a fracture in the same extremity in addition to arterial injury, maximum practicable restoration of arterial circulation is mandatory. The extremity experiences an acute emergency after its circulation has ceased. Normal healing of the fracture implies conditions of circulation aiding the formation of callus and bony consolidation. An extremity with both disturbed arterial circulation and non union is functionally extremely poor.

The surgical approach should in the first place be toward restoration of blood flow. If laceration is concerned the result may be achieved by lateral arterial suture. If the artery is completely transected its ends have to be excised and arteriorrhaphy has to be performed. If the excised segment is of such length that this cannot be done even by mobilizing the artery, the best thing to do is an autogenous vein graft using the vena saphena or an artificial prosthesis. If thrombosis is concerned thrombectomy will

be done. In the cases of arterial spasm in which the artery is collapsed, pulseless and slack, adventitial stripping and local procaine or papaverine injection may produce pulsation. Sympathectomy has a favourable effect in view of the improvement of collateral circulation and may relieve arterial spasm. When the artery is compressed by hematoma it is evacuated and fasciotomy made. In doubtful cases it is advisable to perform arteriotomy in order to reveal a potential distal embolus. The key to success is reestablishment of good flow through the manipulated artery, which is ascertained by distal retrograde flush out manoeuvre using a dilute heparin saline solution. A free flow of saline from the proximal arteriotomy indicates that the vessel is open.

The time lag between the injury and repair of artery is decisive with regard to a successful operation. Degeneration and necrosis of the muscle tissue starts 6-8 hours after arrest of circulation. Arterial repair carried out within this time has the best functional prognosis. Existing collateral paths may prolong this critical period. The necrosis may remain local so that it turns into cicatricial fibrosis and some muscular function remains. Experience has shown that operation is beneficial even when performed at a later time, if the necrosis was not extensive, the extremity will become warmer, the atrophic changes become less and the function is improved. It has been possible to demonstrate also experimentally (12) that there is hope of functional recovery upon nearly total ischaemia is late as after 24 hours. If there is gangrene, however, then amputation is the sole appropriate treatment.

In the treatment of a fracture complicated by arterial injury internal fixation of the fragments has to be considered superior to other methods owing to the primary stability it creates. Especially in fracture of the femur medullary nailing has advantages over other procedures because it prevents redislocation and contributes to secure undisturbed healing of the manipulated vessel. Furthermore, the condition of the extremity can be watched, as it is freely exposed without external supports. If internal fixation cannot be done immediately on account of the time consumed in attending to the arterial injury or for some other reason, the surgery on the bone may be postponed. The essential thing is repositioning of the dislocation and taking care that the fragments cannot compress the artery. Clinical observations show that consolidation of the fracture is distinctly correlated with the arterial circulation of the extremity. If the circulation is deficient, bony consolidation of the fracture is delayed and the result may be fibrous consolidation between the fragments or pseudoarthrosis. It is often indicated to perform early bone transplantation in order to avoid this condition.

## VI DISCUSSION

As a consequence of recent years exigencies in traffic transportation and industry the incidence of severe accidents has increased. As it can be assumed that the number of vascular injuries will increase together with that of multiple traumata avoidance of the loss of extremities and of functional disturbances that are severe even in the best cases seems to require that a more aggressive approach should be adopted towards the restoration of blood flow in the extremity.

With regard to function and viability the development after traumatic interruption of a major artery is decisive (11). The body first responds with a spasm of the vessels in the affected part which might be considered a protective mechanism against the injury. It is partially a reflex constriction mediated through the sympathetic nervous system and partly a traumatic arteriospasm intrinsic in the artery. During this phase of vasoconstriction the circulation of the extremity is most strikingly diminished. The condition of vasodilatation after several hours represents an effort of the organism to increase the blood flow to the part. The point of occlusion in the main artery is bypassed by means of flow through the maximally dilated collateral channels. During the highly unstable period of circulatory readjustment thrombosis is taking place in the occluded artery and it may remain local or spread farther mostly distally. It may occlude the entire arterial tree to the part. The third phase the development of collateral circulation begins following stabilization of thrombosis and vasodilatation. The process takes place fairly rapidly over a period of 4-6 months and then more slowly over a period of years. Usually this collateral circulation never develops the capacity for flow possessed by the original vessel. Residual arterial insufficiency may be apparent as easy fatigue, intermittent claudication, cold intolerance and/or greater susceptibility to the circulatory complications of old age and arteriosclerosis. Where the circulation is sufficiently impaired it results in ischaemic tissue and gangrene. If the initial ischaemia is not too severe and distal thrombosis has not been extensive the extremity may survive the acute occlusion with functional impairment. Besides the site of the lesion has its own significance. It is known that the prognosis is poorest in occlusions of the popliteal and common femoral arteries.

Also as the present work shows acute arterial lesion distally from the aortic bifurcation is dangerous and may develop gangrene especially if both tibial arteries are injured.

The levels of skin demarcation and muscle demarcation may be quite different a fact that carries significance in assessing the viability of the extremity. The nutrient arteries to muscles are frequently end arteries (4). Thus the occlusion may result in death of a muscle or muscle group at much further distal points than might be expected. The present case No. 1 is typical in which the flexor muscles of the leg and quadriceps muscle of a 14 years old boy were necrotic by macroscopic and histologic examination as a result of comminuted fracture of the femur and thrombosis of the femoral artery although good collateral circulation existed. In cases where ischemia is not severe enough to cause massive death the state produces local muscular necrosis. One form is equivalent to the Volkmann's anterior syndrome leading to contracting fibrous replacement of the muscle. If also the nerve is damaged as a consequence of ischemia a combined injury of muscle and nerve is concerned. This occurs characteristically as Volkmann's contracture in the upper extremity but is not rare in the lower extremity either as a sequel of ischemic states and can be demonstrated by muscle biopsy (fig. 11).

The classical symptoms of arterial insufficiency pallor or cyanosis of the skin disappearance of pulse and sensory and motor disturbances are well known. Recognition of a traumatic arterial injury may be more difficult and it may escape observation because the case will mostly be one of a patient under shock and with heavy blood loss whose extremities are cold directly owing to the state of shock and in whom it may be difficult to demonstrate differences in pulse. Also multiple non skeletal trauma cerebral thorax and abdominal injuries may complicate the state to the extent that the symptoms of circulatory insufficiency may fail to receive due attention. But the arterial injury is recognizable by careful clinical examination.

The essential point is that the surgeon taking care of the victim of an accident is minded of arterial lesion as only then diagnosis will be made at once and there is no wastage of valuable time which may decisively affect the chances of saving the extremity. It should be kept in mind that muscular ischemia produced by vascular lesion may be at the bottom of peripheral paresis of the extremity and simulate paralysis caused by the nerve trunk. One must not if there is suspicion of arterial injury wait for it to be possibly repaired of itself but one should verify the diagnosis by arteriography. Too many extremities have been lost by reliance placed on a therapy of waiting. It may furthermore be stressed in this connection that every fracture of an

extremity should be considered to have an associated vascular injury until proved otherwise. One must have the same awareness of the possibility of arterial lesion as one exercises in respect of possible nerve injury, tendon injury or fracture.

## VII SUMMARY AND CONCLUSIONS

This work deals with arterial injuries in connection with fractures of long bones and their surgical approach with the object of restoring the circulation so that the impending loss of the extremity or subsequent atrophy of its function might be avoided.

This investigation comprises only cases verified by arteriography in which injury has produced a critical condition with a view to the viability of the extremity. Of altogether 18 cases four concern injuries of femoral artery, three of popliteal artery, none of anterior and/or posterior tibial arteries, and two concern injury of brachial artery.

The types of acute arterial injury associated with skeletal trauma were: 1) arterial contusion and subsequent traumatic vasospasm, 2) thrombosis, and 3) arterial rupture. The etiology, mechanism and surgical aspects of each type are discussed in the light of the present cases.

The treatment combines two simultaneously applied therapeutic principles, namely those of vascular surgery and bone surgery. The preferential object of treatment is repair of the arterial injury by direct surgery in order to restore the circulation. Treatment of the fracture by medullary nailing has advantages over other methods. In thirteen cases restoration of blood flow by surgical approach was possible and the extremity was saved while in five cases progressive ischaemia had developed so far that the extremity was lost.

The experience shows that in a considerable number of serious fractures with associated arterial injury which would not be considered amenable to surgery by older standards there is a good chance to save the extremity by this approach. Even operation at a later stage is useful unless the ischaemia and necroses are extensive. The coldness of the extremity and the atrophic changes become less and the function of the extremity improves.

*The following more detailed conclusions and general principles emerge from the foregoing presentation:*

*In trauma of an extremity with fracture or dislocation and associated arterial injury, early recognition and appropriate treatment of the latter decide*

*the ultimate fate of the extremity. The surgeon should be minded of arterial injury and until the opposite can be established every fracture of an extremity should be assumed to have associated vascular injury.*

— *Except in cases resulting in serious gangrene the deficiency of active motility, sensory disturbances and contractures of the peripheral parts ensuing on numerous fractures are ischaemic of their aetiology and caused by arterial injury.*

— *By an active arterial surgery it is possible in numerous instances to avoid gangrene and loss or grave functional derangement of the extremity. By accomplishing restoration of circulation in the extremity the indispensable conditions are created for healing of the bone fracture to occur.*

— *Combined arterial injury and bone trauma in an extremity implies combined treatment aiming simultaneously at restoration of blood flow and bony consolidation of the fracture in this order. Of course all necessary therapeutic measures should be taken to save the patient's life without harding anything even if this be at the risk of loss of the extremity. In multiple injuries observance of the proper order of preference in therapy is the fundamental prerequisite of success.*

— *Treatment of the fracture by medullary nailing has advantages over other methods in that it guards against redislocation and thus contributes to ensuring undisturbed healing of the surgically manipulated artery. Another advantage is the possibility of watching the condition of the extremity unobstructed by external supports.*

## VIII ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

Die vorliegende Arbeit befasst sich mit Arterienverletzungen im Zusammenhang mit Frakturen und Luxationen und mit ihrer chirurgischen Behandlung im Sinne der Wiederherstellung des Blutlaufs um den vorauszuwendenden Verlust der betroffenen Extremität bzw. schwergradig gestörte Funktion derselben vermeiden zu können.

Die Untersuchung umfasst ausschliesslich arteriographisch erhaltete Fälle, in denen die Verletzung einen kritischen Zustand im Hinblick auf die Erhaltung der Extremität herbeigeführt hat. Von den insgesamt 18 Fällen beziehen sich vier auf Verletzungen der A. femoralis, drei auf solche der A. poplitea, neun auf solche der A. tibialis anterior und/oder posterior und zwei auf eine Verletzung der A. brachialis.

Folgende Typen der Arterienverletzung in Verbindung mit Knochen- trauma kamen vor: 1) Arterienkontusion und anschliessender Vasospasmus, 2) Thrombose sowie 3) Riss der Arterie. Die Ätiologie, der Mechanismus und die chirurgischen Aspekte eines jeden dieser Typen werden im Lichte der hier berichteten Fälle besprochen.

In der Behandlung vereinigen sich zwei simultan in zum Einsatz kommende therapeutische Prinzipien, die der Gefässchirurgie und der Knochenchirurgie. Das therapeutische Ziel mit Vorrang ist Instandsetzung der verletzten Arterie durch unmittelbaren chirurgischen Eingriff zum Zweck der Wiederherstellung des Kreislaufs. Behandlung der Fraktur mittels Marknagelung besitzt Vorteile den anderen Verfahren gegenüber. Sie gewährleistet einen Schutz gegen Rediszlokierung und trägt ihrerseits zum Sichern einer ungestörten Heilung der Arterie bei. In 13 Fällen wurde Wiederherstellung des Blutlaufs durch chirurgischen Eingriff erzielt und die Extremität gerettet, während in fünf Fällen die progressive Ischämie so weit fortgeschritten war, dass die Extremität amputiert wurde.

Die Erfahrungen erweisen, dass in einer grossen Zahl von ernsthaften Frakturen mit assoziierter Arterienverletzung, die nach älteren Auffassungen nicht als chirurgisch bezwingbar angesehen wurden, gute Möglichkeiten bestehen, die Extremität durch diese Behandlungsweise zu retten. Selbst erzielte Operation ist von Nutzen, insofern nicht die Ischämie und die



Nekrosen grosse Ausdehnung aufweisen die Kälte der Extremität und die atrophischen Veränderungen werden geringer und bessere Funktion der Extremität stellt sich ein

Die folgenden mehr ins Einzelne gehenden Folgerungen und allgemeinen Richtlinien ergeben sich aus der vorangehenden Darstellung

— Bei Extremitätentrauma mit Fraktur oder Luxation und assoziierter Arterienverletzung ist zeitiges Erkennen und sachgemasse Behandlung der letzteren entscheidend hinsichtlich des bevorstehenden Schicksals der Extremität. Der Arzt soll sich der Arterienverletzung bedacht sein bis Gegen teiliges nachgewiesen ist. Ist jede Fraktur als von einer Arterienverletzung begleitet anzunehmen

— Ausser in Fällen die zu schwerartigem Gangrän führen sind mangel hafte aktive Beweglichkeit sensorische Störungen und Kontrakturen der peripheren Teile ischämischer Ätiologie und von einer Arterienverletzung verursacht

— Durch aktives therapeutisches Vorgehen mit der Wiederherstellung des Blutkreislaufs in der Extremität mit Knochentrauma und Arterien verletzung als Ziel können in zahlreichen Fällen Gangrän und Gliedmassenverlust bzw. schwere funktionale Beeinträchtigung vermieden werden. Durch Wiedereingangbringen der Extremitätenzirkulation schafft man die unerlässlichen Vorbedingungen für die Heilung der Knochenfraktur

— Kombinierte Arterienverletzung und Knochenfraktur in einer Extremität setzt eine kombinierte Therapie voraus die zugleich auf Wiederher stellung des Blutlaufs und auf knocherner Konsolidation der Fraktur hin zielt und zwar in dieser Reihenfolge. Es versteht sich dass alle zur Lebens rettung des Patienten notwendigen therapeutischen Massnahmen an erster Stelle vorzunehmen sind ohne irgend etwas aufs Spiel zu setzen sogar auf Gefahr eines eventuellen Gliedmassenverlusts. Bei multiplen Traumata ist Beachtung einer richtigen Vorrangordnung in der Therapie Grundbe dingung für den Erfolg

— Behandlung der Fraktur mittels Marknagelung ist anderen Methoden gegenüber im Vorteil indem sie einer Redislozierung vorbeugt und dadurch zur ungestörten Heilung der chirurgisch manipulierten Arterie beiträgt. Ein weiterer Vorzug ist die Möglichkeit zum Verfolgen des Zustands der Extremität ohne Behinderung durch ausserliche Fixierstützen

## IX. ACKNOWLEDGEMENTS

The author feels great pleasure in expressing his gratitude towards Professor K. I. Kulho M.D. Chief of the Clinic for Orthopaedics and Traumatology, University of Helsinki, whose kind attitude and valuable advice have been a source of inspiration and encouragement to my interest in the subject of this work.

I am obliged to A. Hailonen M.D. for his fine arteriograms. I also wish to mention my other colleagues in the Clinic, as well as its nurses, in sincere appreciation of their help and contribution.

Mr. U. Attila M.Sc. who has translated the manuscript, is deserved of my best thanks.

## X REFERENCES

- 1 *De Bakey M E and Simeone F I* Battle Injuries of the Arteries in World War II — *Ann Surg* 534 1 3 1946
- 2 *Cnley J E and Boulanger W J* Management of Arterial Injuries to Lower Extremity — *Wisconsin Med J* 1960 59 185
- 3 *Edwards E I* The Anatomic Basis for Ischemia Localized to Certain Muscles of the Lower Limb — *Surg Gyn Obst* 1953 97 87
- 4 *Hoover A H* Injuries of the Popliteal Artery Associated with Fractures and Dislocations — *Surg Clin North America* 1961 11 1099
- 5 *Jahnke Jr E J* Late Structural and Functional Results of Arterial Injuries Primarily Repaired — *Surgery* 1958 43 175
- 6 *Jahnke F J Jr and Howard J M* Primary Repair of Major Arterial Injuries — *AMA Arch Surg* 1953 86 646
- 7 *Jahnke F J Jr and Seeley S F* Acute Vascular Injuries in the Korean War — *Ann Surg* 1953 138 158
- 8 *Koskinen E I S* Arterial Injuries Leading to Gangrene as Complications of Fractures of the Lower Extremities — *Ann Chir et Gynaec Fenn* 1958 47 99
- 9 *Jeriche R* Surgery of Sympathetic System: Indications and Results — *Ann Surg* 1928 88 449
- 10 *Jinden L W F* Recent Progress in Peripheral Arterial Surgery — *Ann Chir et Gynaec Fenn* 1961 60 Suppl 99
- 11 *Linton R R, Shattuck R S and Donaldson G I* Blood Vessel Injuries in Fractures and Other Injuries. Editor E F Cave — The Year Book Publishers Inc Chicago 1958
- 12 *Miller H H and Welch C S* Quantitative Studies of the Time Factor in Arterial Injuries — *Ann Surg* 1949 130 428
- 13 *Spear H C and Janes J M* Rupture of the Brachial Artery Accompanying Dislocation of the Elbow or Supracondylar Fracture — *J Bone & Joint Surg* 1951 33 1 883
- 14 *Stein A H Jr* Arterial Injury in Orthopaedic Surgery — *J Bone & Surg* 1956 38 1 669
- 15 *Torup D* Arterielæsioner i tilslutning til frakturer og frakturbehandling — *Nord Med* 1961 66 111
- 16 *Watson Jones R* Fractures and Joint Injuries Vol I — E & Livingstone Ltd Edinburgh and London 1955
- 17 *West J P, Winant F M and Blair C R* Femoral Artery Reconstruction in a Patient with a Compound Fracture of the Femur — *J Bone & Joint Surg* 1951 39 1 139 4







ACTA ORTHOPÆDICA SCANDINAVICA  
SUPPLEMENTUM No LVIII (58)

---

FROM THE CLINIC FOR ORTHOPÆDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI

CHIEF PROFESSOR K E KALLIO

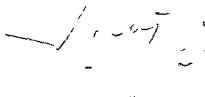
AND THE DEPARTMENT OF FORENSIC MEDICINE UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR U. UOTILA

RÔLE OF SURGICAL INTERVENTIONS  
OF THE HIP JOINT IN THE AETIOLOGY OF  
ASEPTIC NECROSIS OF THE FEMORAL HEAD

EXPERIMENTAL STUDY

BY

PENTTI ROKKANEN



MUNKSGAARD  
COPENHAGEN 1962

345  
174





ACTA ORTHOPÆDICA SCANDINAVICA  
SUPPLEMENTUM No LVIII (58)

---

FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR A. E. KALLIO  
AND THE DEPARTMENT OF FORENSIC MEDICINE UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR U. UOTILA

RÔLE OF SURGICAL INTERVENTIONS  
OF THE HIP JOINT IN THE AETIOLOGY OF  
ASEPTIC NECROSIS OF THE FEMORAL HEAD

EXPERIMENTAL STUDY

BY  
PENTTI KOKKANEN

---

MUNKSGAARD  
COPENHAGEN 1962



RÔLE OF SURGICAL INTERVENTIONS  
OF THE HIP JOINT IN THE AETIOLOGY OF  
ASEPTIC NECROSIS OF THE FEMORAL HEAD



ACTA ORTHOPÆDICA SCANDINAVICA  
SUPPLEMENTUM No LVIII

---

FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI

CHIEF PROFESSOR L. E. KALLIO

AND THE DEPARTMENT OF FORENSIC MEDICINE UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR U. UOTILA

RÔLE OF SURGICAL INTERVENTIONS  
OF THE HIP JOINT IN THE AETIOLOGY OF  
ASEPTIC NECROSIS OF THE FEMORAL HEAD  
  
EXPERIMENTAL STUDY

BY  
PENTTI ROKKANEN

---

MUNKSGAARD  
COPENHAGEN 1962



ACTA ORTHOPÆDICA SCANDINAVICA  
SUPPLEMENTUM No LVIII

---

FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR K E KALLIO  
AND THE DEPARTMENT OF FORENSIC MEDICINE UNIVERSITY OF HELSINKI  
CHIEF PROFESSOR U UOTILA

RÔLE OF SURGICAL INTERVENTIONS  
OF THE HIP JOINT IN THE AETIOLOGY OF  
ASEPTIC NECROSIS OF THE FEMORAL HEAD

EXPERIMENTAL STUDY

BY  
PENTTI ROKKANEN

---

MUNKSGAARD  
COPENHAGEN 1962





## PREFACE

The subject of this work was suggested by my highly respected teacher and present principal, Professor K. E. KALLIO M D, Chief of the First Surgical Clinic of the Central Hospital, University of Helsinki the Clinic for Orthopaedics and Traumatology and Surgeon in Chief of the Toolo Hospital. He has not spared his advice, encouragement and constructive criticism in the different stages of my work, and I am very happy of this opportunity to express my deep gratitude for his attention.

The microscopic work and the special investigations involved in my studies were carried out in the Department of Forensic Medicine University of Helsinki. Its Chief, my former teacher Professor U. UOTILA M D, has given me most valuable aid in the form of ample advice and unflinching criticism. My sincerest thanks are due to him for this and for the use of the Department's facilities.

The interest shown towards my work by University Lecturer R. KOHLER M D, has enabled me to make the required x rays in the Roentgenological Department of the Surgical Clinics of the University of Helsinki. My inclination towards scientific research was first inspired by University Lecturer S. VAINIO M D, at the time I was working under him. I feel greatly obliged to them both.

The English translation of my manuscript was made by Mr U. ATTILA, M Sc, and read by Miss ELVI KALLOKALLIO. In the compilation of references from the pertinent literature I have been aided by Miss AINO NIKU PAANO M A, and by Mrs. EENA KJILBERG M A. Assistance in the technical aspects of the work was given by Mr J. SALORANTA M Sc in Eng, who also drew the diagrams reproduced here. Mr H. LAINE B M S assisted me in the operations on the rabbits and in the x ray work, and Mr A. TAMMINE in the microradiographic technique. The histologic preparations were processed by Mrs. LISBETH SCHWANCK, and Mr P. KORHONEN of the Photographic Department of the University of Helsinki made the micrographs. All these persons are deserved of my sincere thanks.

The operation room nurses of the First Surgical Clinic have placed at my disposal the equipment and materials needed in the operations, while

the nurses of its Roentgenologic Department assisted me in the x ray procedure I wish to express my gratitude to them as well as to all others not specifically mentioned here who have aided my work in various ways

To my wife AILA ROKKANEN Lic Med, I am deeply thankful for her loving help and understanding throughout the progress of the work

This work has been partially aided by an institutional grant from the SIGRID JUSÉLUS Foundation Helsinki to the Department of Forensic Medicine University of Helsinki

Helsinki May 1962

*Pentti Rokkanen*

## CONTENTS

|  | Page |
|--|------|
| INTRODUCTION   | 9    |
| General Considerations   | 9    |
| Some Observations on the Blood Supply of Bone and Its Disturbances                 | 9    |
| Some Special Investigations for Clarification of the Blood Supply of Bone          | 13   |
| PROBLEMS   | 17   |
| MATERIAL AND METHODS   | 18   |
| Experimental Animals and Operation Techniques                                      | 18   |
| Methods of Examination   | 22   |
| ARTHROTOMY DIVISION OF LIGAMENTUM TERES AND TEMPORARY LUXATION OF THE FEMORAL HEAD | 25   |
| Earlier Investigations   | 25   |
| Own Investigations   | 26   |
| Comments   | 34   |
| ARTHROTOMY AND LUXATION WITHOUT REDUCTION  | 36   |
| Earlier Investigations   | 36   |
| Own Investigations   | 39   |
| Comments   | 46   |
| ARTHROTOMY AND CAPSULECTOMY  | 50   |
| Earlier Investigations   | 50   |
| Own Investigations   | 52   |
| Comments   | 55   |
| ARTHROTOMY AND NEARLY TOTAL OSTEOTOMY OF THE NECK OF THE FEMUR                     | 57   |
| Earlier Investigations   | 57   |
| Own Investigations   | 59   |
| Comments   | 67   |
| ARTHROTOMY AND TIGHT LIGATION OF THE NECK OF THE FEMUR WITH STEEL WIRE             | 70   |
| Earlier Investigations   | 70   |
| Own Investigations   | 71   |
| Comments   | 82   |
| LIGATION OF THE DEEP FEMORAL ARTERY AND THE OBTURATOR ARTERY                       | 85   |
| Earlier Investigations   | 85   |
| Own Investigations   | 87   |
| Comments   | 88   |
| DISCUSSION   | 89   |
| SUMMARY AND CONCLUSIONS  | 95   |
| References   | 99   |



## INTRODUCTION

### GENERAL CONSIDERATIONS

Regardless of the method that may be applied arthroplasty of the hip joint involves opening of the joint division of the ligamentum teres dislocation of the femoral head and even capsulectomy It is only natural that in a clinic which applies its own method skin arthroplasty, and in which much reconstructive work is also otherwise done on the hip joint there is a desire to penetrate into fundamental factors for such surgical interventions Accordingly I was advised in the autumn of 1958 by my present principal Professor K. E. KALLIO M.D. to find out by means of animal experiments whether the above mentioned operations might produce aseptic necrosis of the bone of the femoral head In the course of the work it turned out to be worth while to investigate whether the cancellous circulation might also be affected by other expedients This is the background against which the present work has taken shape

### SOME OBSERVATIONS ON THE BLOOD SUPPLY OF BONE AND ITS DISTURBANCES

*Clinical investigations* — An early observation concerning the circulation of the femoral head was made by HUNTER (1743) who found that the blood vessels prior to entering the bone are anastomosed in the subsynovial layer constituting the so called *circulus articuli vasculosus* Numerous investigations (PALLETTA 1820 COOPER 1823 SAPPEY 1869 LANGER 1875) were carried out in the last century also concerning the blood supply of the femoral head although they were mainly macroscopic in character with no tracing of the circulation within the bone in greater detail being done LANGER (1875) already showed that bones have their main arteries but awareness of the circulatory disturbances of bones and joints was slow in developing It is certainly true that VOLKMANN (1864) described multiple suppurating infarcts in bones in connection with septic endocarditis KONIG (1888) characterized osteochondritis dissecans as an independent disease attributing it to contusion with a subsequent necrotic

process and inflammatory sequestration of a bone fragment, but he was not able to explain the origin of the necrosis. The first observation relating to aseptic disturbance of circulation of the bone was made in association with bone transplantation. This was when BARTII (1893) observed that the transplanted bone tissue dies and is later replaced with new bone.

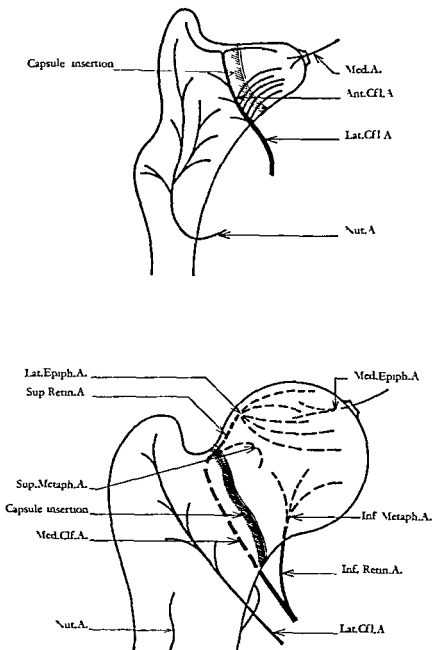
Aseptic necrosis of bone will occur in a place where the blood supply of the bone has been disturbed to a sufficient degree. It is most readily produced in the epiphysis of the bone. Accordingly the most numerous observations in earlier times were made in connection with so called spontaneous epiphyseal necrosis. Here, we have reason to mention AX. HALSEN who has studied the bone necrosis problem already since 1911 and who created in 1922 the actual concept of avascular necrosis of the bone by clarifying its histologic picture and combining the concepts of osteochondritis dissecans and spontaneous epiphysitis. He described in a patient aged 16 necrosis of the bone after a ten months old fracture of the neck of the femur, in which he found total necrosis of the spongy bone, bone marrow and epiphyseal cartilage, and partial necrosis of the articular cartilage.

It has been realized later that traumata, such as fracture of the neck of the femur and dislocation of the femoral head may cause necrosis of the bone on the basis of circulatory disturbance. Also contusion may result in necrosis of the bone. According to PREMISTIR (1931) JACKSON and BICKELL (1948) and BLOUNT (1956) necrosis of the bone subsequent to fracture of the trochanter is rare. Furthermore necrosis of the bone is encountered in the caisson disease (*e.g.* McCALLUM *et al.* 1954) and in certain forms of anaemia (*e.g.* TANAKA *et al.* 1956). Moreover BOYD (1957) has described the so called coronary disease of the hip.

Necrosis of the bone occurs also in connection with arteriosclerosis. For instance MULLER (1927) observed in four, JAFFE and POMERANZ (1934) in seven and SHERMAN and SELAKOVICH (1957) in ten patients subjected to amputation on account of circulatory disturbance a deranged blood supply and necrosis of the bone particularly in the intermediate lamellae. Also in connection with normal aging arteriosclerosis and thus necrosis of the bone occurs physiologically already, just as connective tissue degenerates as it becomes older (RISSANEN 1960).

We thus see that the states are numerous in which aseptic necrosis of the bone plays a central part in orthopaedic surgery even apart from the bone necrosis phenomenon encountered in connection with normal repair of a fracture. In his extensive monograph HAUPTLI (1954) has reviewed the chondro-osteonecroses.

*Fig. 1 Anterior view of the blood supply of the femoral head in adult rabbit and man*



TRUETA and HARRISON (1953) and after them JUDET *et al* (1955) in respect of adults, and TRUETA (1957), in respect of children were the first to clarify the blood supply in the femoral head and neck in persons of different age and to show that it changes in the course of time Fig 1 is a schematic presentation of this blood supply in the femoral head of a human adult according to TRUETA, and, for comparison that in the head of the rabbit femur according to LEMORNE (1957) Not until this normal anatomy was clarified was a basis established for investigation of circulatory disturbances and necrosis of the bone and for their understanding in various clinical conditions relating to the head of the femur

*Animal experiments* — Numerous experiments have been arranged to produce necrosis of the bone in animals and to clarify its clinical and histological picture CORNIL and COUDRAY (1904) showed that in bone fractures in the rabbit necrosis of the cortex ensues at some distance from the line of fracture and that this is followed by replacement of the dead bone with new bone The importance of the epiphyseal line was pointed out by an observation made by JACKSON (1913) When he attempted to induce experimental arthritis in rabbits by intravenous injection of pyrogenic organisms wedge like infarcts of bone were produced which were located in the metaphyses immediately under the epiphyseal cartilage This indicated that the main arteries of the epiphysis may be obstructed by emboli Later investigations have also shown that the epiphysis may act as an obstruction preventing blood flow to the head of the bone (HARRIS and HOBSON 1956)

ANHAUSEN (1914) produced necrosis of the bone by application of heat cold and tincture of iodine ELOESSER (1917) observed that sectioning of a nerve of a certain kind without sympathectomy produced articular changes resembling Charcot's disease BANCROFT (1922) introduced croton oil into the dog's humerus by intramedullary injection obtaining necrosis and rapid involucrum formation as well as swift healing He stressed the importance of the blood supply in bone regeneration Using radium PHEMISTER (1926) produced necrosis of bone in the humerus and knee joint of dogs BRUNSCHWIG (1930) found that detachment of the periosteum in young dogs caused extensive necroses in the femur and tibia In the head of the metatarsus of a growing dog SACERDOTE (1931) produced marked necrosis and deformity by detaching the periosteum from the fractured segment Either removal of the periosteum or fracture resulted in a much lesser degree of vascular changes and attendant necroses

CORBIN (1937) studied the question in experiments with 15 cats, finding that section of lumbal and sacral nerve roots produced considerable ar-



thritic changes although also sympathectomy had been performed the cartilage degenerated was destroyed and the spongiosa became sclerosed THOMSEN (1939) investigated the humerus and femur of six months old pigs weighing about 66 kg he found changes in five bones out of 2203 of the latter kind and in 62 out of 1482 (4 %) of the former They were resemblant of infarct situated subchondrally with the base towards the cartilage Microscopic examination revealed fibrous tissue abundant cells and new blood vessels in the cancellous spaces but no signs of osteoblasts or osteoclasts nor any of actual necrosis All the same, he considered the changes most nearly like those of *osteochondritis juvenilis*

Remarkable were also the clinical as well as experimental studies carried out by NAGURA (*e.g.* 1937 1938, and NAGURA and KOSUGE 1938) in his investigation of the similarity in aetiology between osteochondritis dissecans and so called spontaneous epiphyseal necrosis With a hammer blow he induced a blunt trauma at the upper and lower ends of the femur in young rabbits and followed the resulting changes histologically, in addition to the immediately ensuing demolition and rebuilding process also another secondary such process took place (the so called phenomenon of NAGURA) According to him, in every avascular bone necrosis the primary occurrence is a small subchondral fracture, and the bone necrosis is produced traumatically BURCHARDT (1948) repeated NAGURA's work with rabbits of 5—7 weeks age confirming his findings

RUTISHAUSER and MAJNO (1949) resected part of the radius in dogs producing osteonecrosis in the ulna, on the basis of an overexertion phenomenon The same investigators (RUTISHAUSER and MAJNO 1951) also paid increased attention to the changes of various kinds occurring in the osteocyte itself and in the ground substance in conditions of bone necrosis TRUETA (1954) performed experimental investigations of the regeneration of bone in rabbits and found that the presence of dead bone was important though not indispensable in order that regeneration of the bone might take place The sequel of the reaction caused by dead bone was hyperaemia according to HARRISON *et al* (1953)

#### SOME SPECIAL INVESTIGATIONS FOR CLARIFICATION OF THE BLOOD SUPPLY OF BONE

By the usual clinical means futile attempts have been made to elucidate the circulation of the femoral head in various traumatic conditions

At roentgenologic assessment of avascular necrosis of bone one has to observe according to the clinical and experimental work of LACHMAN and WHELAN (1936) that the loss of calcium must amount at least to 20 per cent before the change in density becomes obvious in the x ray

According to WATSON JONES and ROBERTS (1934) increased density in an avascular bone is merely a secondary phenomenon when the adjacent living bone is hyperaemic and decalcified. This occurs because calcium cannot be mobilised from the dead fragment and the density therefore remains unchanged. Admittedly many investigators are of the opinion that the bone is not really dead but that rather there occurs transient reossification with new bone being apposed on the surface of the dead bone trabeculae, producing so called creeping substitution (*e.g.* PHEMISTER 1935, 1939 DE HAAS and MACNAB 1956 and BONFIGLIO and BARDENSTEIN 1958).

Thus bone may in likeness with other dead tissues take up calcium salts from its surroundings and the density will increase also by this mechanism. This provides indeed an explanation for the trabecular structure and for the increased density at the point in question in the x rays. Moreover it should be noted that ossified intercellular matter does not change immediately even in the roentgenologic respect. As ALHAUSEN (1922) has observed dead bone (the avascular organic matrix) gradually loses its strength and resistance so that it will collapse and the density will in fact increase. BOBECHO and HARRIS (1960) observed in the course of repair of bone necrosis experimentally induced in rabbits that in the fifth week the new cancellous cavities were smaller in size in comparison with those existing before and the density was thus indeed due to reossification. They furthermore corroborated their result by autoradiography with radio calcium. They also reported that the density began to be observable at 3 weeks, but with certainty only at 8 weeks after inception of the bone necrosis. Since in man the corresponding change takes place mostly at an even slower rate it is evident that this method too, is slow and judgment of its results is difficult.

As a means to obtain results more rapidly, arteriography has been applied by, *e.g.* ROOK (1953) and MUSSBICHLER (1956) in attempts to clarify the blood supply of the femoral neck in fractures. However the investigation renders mainly a picture of the circulatory disturbances that occur outside the bone. Microangiography (BARCLAY 1951) has not been developed for elucidation of man's intraosseal circulation in clinical conditions. HULTH (1956 1958 1958) suggested venography as a method by which in connection with operation an idea of venous damage and indirectly also of trauma to the parallel artery can be obtained, and he considered that in this manner the extent of the avascular bone necrosis to be expected might be inferred. BENASSY and LAMARE (1956) for instance have also employed venography in their investigations on coxarthroses.

There have been attempts to clarify the blood supply of bones with the aid of radioactive isotopes. In England TUCKER (1950) was the first to apply radio phosphorus clinically after preliminary tests with rabbits in order to determine the circulation in the femoral head in 13 patients who had a fractured neck of the femur. Similarly in a series of 45 patients with fractured neck of the femur ARDEN and VEALL (1953) took bone specimens from the femoral head and from the trochanteric region, examining their radioactivity and comparing the relative values indicating the blood supply of the femoral head. They considered their results encouraging. BORD *et al* (1955) investigated this question with 15 dogs in which avascular necrosis had been produced by division of the ligamentum teres inflicting damage to the capsule and furthermore performing in some instances osteotomy on the neck of the femur. In addition they studied 53 clinical cases. LAING *et al* (1957) administered radio phosphorus to ten rabbits by intravenous injection and took bone specimens at different points. They stressed the importance of the time when the specimens are taken. LAING and FERGUSON (1959) produced in 33 dogs dislocation of the femur in connection with damage caused to the lig. teres and to other blood vessels. In addition they studied avascular bone necrosis with the aid of radioactive sodium. They were well satisfied with their results.

In contrast with the preceding report ARDEN (1960) after having assembled 100 clinical cases with an observation period exceeding two years noted a primary error percentage of 40 per cent, while one fifth of the remaining results were doubtful. He placed weight on the significance of the point of measurement that is on its equivalent location, because the radio activity is high close to the lig. teres owing to its still active circulation while it is low in the neighbourhood of the fracture itself. He considered additional studies to be necessary before the method might be adopted for general use. Also STEVENS (1960) arrived at a similar result in his experiments with rats. NORDBY (1960) reported the successful use of radioactive iodine to establish unilateral cases of necrosis of the femoral head. In experimental studies autoradiography with various isotopes has also been employed as a means to clarify the vascularization of bone as has been described by FITZGERALD and ENGSTROM (1952) and by BORD (1955). However owing to their inconvenience the methods have not found their way into everyday clinical usage.

HULTH (1961) employed microradiography in four cases in his investigation of aseptic necrosis of the femoral head. He said that the trabecular bone structure was well preserved and that the hypersclerosis was due to revitalization of bone.

According to WATSON JONES and ROBERTS (1934) increased density in an avascular bone is merely a secondary phenomenon when the adjacent living bone is hyperaemic and decalcified. This occurs because calcium cannot be mobilised from the dead fragment and the density therefore remains unchanged. Admittedly, many investigators are of the opinion that the bone is not really dead but that rather there occurs transient reossification with new bone being apposed on the surface of the dead bone trabeculae producing so called creeping substitution (*e.g.* PHEMISTER 1935 1939 DE HAAS and MACNAB 1956 and BONFIGLIO and BARDENSTEIN 1958).

Thus bone may in likeness with other dead tissues, take up calcium salts from its surroundings and the density will increase also by this mechanism. This provides indeed an explanation for the trabecular structure and for the increased density at the point in question in the x rays. Moreover it should be noted that ossified intercellular matter does not change immediately even in the roentgenologic respect. As ALHAUSEN (1922) has observed, dead bone (the avascular organic matrix) gradually loses its strength and resistance so that it will collapse and the density will in fact increase. BOBECHO and HARRIS (1960) observed in the course of repair of bone necrosis experimentally induced in rabbits that in the fifth week the new cancellous cavities were smaller in size in comparison with those existing before and the density was thus indeed due to reossification. They furthermore corroborated their result by autoradiography with radio-calcium. They also reported that the density began to be observable at 3 weeks but with certainty only at 8 weeks, after inception of the bone necrosis. Since in man the corresponding change takes place mostly at an even slower rate it is evident that this method too, is slow and judgment of its results is difficult.

As a means to obtain results more rapidly, arteriography has been applied by *e.g.* ROOK (1953) and MUSSBICHLER (1956) in attempts to clarify the blood supply of the femoral neck in fractures. However, the investigation renders mainly a picture of the circulatory disturbances that occur outside the bone. Microangiography (BARCLAY 1951) has not been developed for elucidation of man's intraosseal circulation in clinical conditions. HULTH (1956 1958 1958) suggested venography as a method by which in connection with operation an idea of venous damage and indirectly also of trauma to the parallel artery can be obtained and he considered that in this manner the extent of the avascular bone necrosis to be expected might be inferred. BENASSY and LAMARE (1956) for instance have also employed venography in their investigations on coxarthroses.

## PROBLEMS

*The purpose of the present work was to elucidate the following questions associated with arthrotomy*

1 What signs of aseptic necrosis either macroscopic or roentgenologic are produced in the femoral head by arthrotomy of the hip joint division of the lig. teres and temporary luxation of the femoral head in rabbits 6—7 months of age?

2 What changes are produced in the femoral head when it has been subjected to similar dislocation by operative interference and subsequently left in a luxated state?

3 What changes are produced in the femoral head by arthrotomy and capsulectomy

4 What changes are produced in the femoral head by nearly total osteotomy of the neck of the femur?

5 What changes are produced in the femoral head by tight ligation of the neck of the femur with steel wire

6 What changes are produced in the femoral head by the mere ligation of the deep femoral artery and the obturator artery?

7 Does such aseptic necrosis as may be called forth by the above mentioned interferences present a correlation with the changes in the articular cartilage

8 How is aseptic necrosis of bone demonstrated histologically, autoradiographically and microradiographically?

We may thus state that it has not been possible so far to observe any positive signs of necrosis of the bone other than the lack of nucleus in the osteocyte which was demonstrated by ANHALSEN (1922 1926 1928) and by ANHALSEN and BERGMANN (1937) and which they considered absolutely reliable. It is certainly true that the phenomenon takes place at a slow rate. Although the bone can be considered dead already 4—5 days after its blood supply has been arrested, disappearance of the nucleus in the osteocyte does not occur until after 2—3 weeks as was shown by WOLLENBERG (1928) in his experiments with white mice. The increase of fibrous tissue in the cancellous spaces furnishes an indication of incipient regeneration and subsequent metaplasia, with simultaneous replacement of the dead bone by new bone—a phenomenon which PHEMISTER (1926) has named creeping substitution as stated above.

Chemicals and dyes, such as *e.g.* fluorescein have also been employed for the purpose of demonstrating necrosis of the bone but the methods have been rejected because of failure to elicit sensitive response.

The facts emerging from this review seem to justify another attempt at clarification by means of animal experiments of questions relating to the aetiology of aseptic necrosis of bone.



The operations were performed in the operating room of the clinic animal stables under careful asepsis. An undiluted solution «Diminal» of Astra (Sweden) was used as anaesthetic. During operation the animal was kept lying freely on its right side with no restraining bands. From the area to be operated, the hairs were cut with scissors and it was cleaned with «Iodopax» of Ferrosan (Sweden).

The left hip was explored through a posterolateral incision following the course of the musculature. The posterior surface of the joint was reached after the sciatic nerve had been pulled to one side. The joint capsule was opened in all other cases except in those of Group VI.

Thereafter, in Group I, the ligamentum teres was divided after the joint space had been rendered more wide by pulling the extremity ventrally and rotating it inwards. The femur was luxated backwards and kept in this position for a while after which it was repositioned in the acetabulum. The capsule was not sutured but the margin of the muscle was drawn up to cover it and the wound was closed by layers with thin catgut. Two separate applications of «Nobecutan» plastic film spray of Bofors (Sweden) were made in the sutured wound.

In Group II the extremity was left permanently in its luxated position.

In Group III the operation described above was supplemented with capsulectomy during the phase of luxation which invariably resulted in heavy haemorrhage from the ventral capsule.

In Group IV osteotomy of the neck of the femur was performed in connection with the luxation applying an electric saw so that the osteotomy surface was equivalent to at least two thirds of the diameter of the neck. This osteotomy was made in the middle of the neck of the femur starting from its caudal margin. The femoral head was then repositioned and the wound closed in the usual manner.

In Group V the neck of the femur was ligated with a tightly applied steel wire while the femoral head was held luxated. For attachment of the wire the greater trochanter was pierced with an electric drill and the end of the steel wire was passed through the hole. The steel wire thus formed a double loop.

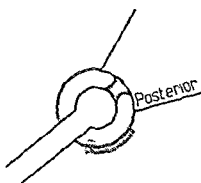
In Group VI the joint was not entered at all but the large vessels which branch off later into the joint, i.e. the deep femoral artery and the obturator artery were ligated. During this operation the muscles attaching to the greater trochanter were cut off temporarily.

The different operations that were performed are shown in the schematic diagrams in Fig. 2.

Each rabbit received postoperatively 100,000 I.U. of procaine peni-



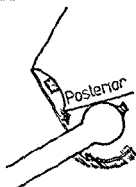
Fig 2 Operations performed



## GROUP I

## Arthrotomy

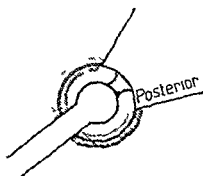
Division of ligaments Temporary luxation  
of the femoral head



## GROUP II

## Arthrotomy

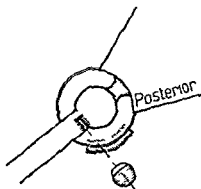
Luxation of the femoral head without  
reduction



## GROUP III

## Arthrotomy

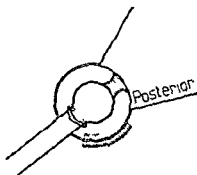
## Capsulectomy



## GROUP IV

## Arthrotomy

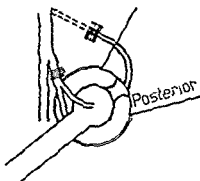
Nearly total osteotomy of the neck of the  
femur



## GROUP V

## Arthrotomy

Tight ligation of the neck of the femur  
with a steel wire



## GROUP VI

Ligation of the deep femoral artery and the  
obturator artery

cillin intramuscularly and was transferred to its cage while still under anaesthesia. In one instance distinct infection was observed in the joint and another animal was operated to replace the subject.

The individual rabbits were marked according to a systematic key in which for instance the notation G 15 refers to the animal treated in Group I and sacrificed on the fifth postoperative day.

#### METHODS OF EXAMINATION

The experimental animals were put to death by injection into the ear vein of a lethal dose of an anaesthetic intended for intravenous use.

Visual observations were made when both hip joints were being exposed and the femoral heads were being excised.

As a rule two x-ray examinations were made of each experimental animal, one being done a few days after the operation and the other immediately after sacrificing. If the animal was scheduled to be sacrificed on the 5th to 15th postoperative day, only the latter x-ray was made. For x-raying the rabbit was placed in a special fixture and strapped down by its trunk. The hind legs were held by hand in a symmetrical position while the exposure was made on non-screen «Kodirex» (Kodak) film with the tube at 2 m distance and settings at 105 mAs and 65 kV. In the x-rays the operated and the intact hip were compared as well as the status with respect to radiologic density of the bone after operation and after sacrificing. Attention was also paid to narrowing of the joint space, to establishment of a new socket in the luxated cases, and to changes in the pelvic bones.

Both femoral heads of the rabbit, including part of the neck, were taken as specimens for histological examination. The specimens were fixed in neutral 10 per cent formalin and decalcified with 32 per cent (vol/vol) EDTA (ethylene diamine tetra acetic acid disodium salt) solution of pH 7.2—7.4 at about 37°C. Upon decalcification the samples were dehydrated by passing them through a series of baths with increasing alcohol content (50, 70, 80, 95 per cent and absolute alcohol) and transferred through benzene into paraffin in the usual manner. Subsequently they were kept in paraffin at about 63°C in an incubator for about one week. This is a good though slow procedure.

Sectioning was done with a standard microtome. The femoral head was split through the middle in the frontal plane so that the ligamentum teres fell into the plane of cleavage. According to the histologic findings this was successful in all instances. The basic stain was Weigert van Gieson's haematoxylin. In addition Pentachrome staining was used.

Pentachrome I and II staining (Movar 1955) is a method by which all connective tissue elements also the acid mucopolysaccharides of the ground substance can be made visible in one and the same section. It is a combined staining method composed of Weigert's iron haematoxylin for nuclei, woodstain scarlet acid fuchsin or orange G for cytoplasm, resorcin fuchsin for elastic tissue, saffron or diphenyl fast red for collagen and Alcian blue for the ground and cementing substances. The bone acquires an intensive red stain with Pentachrome II, hyaline cartilage a bluish or blue green stain and fibrous cartilage a greenish yellow or brown stain. Fibrous tissue is stained red or brownish red, the cytoplasm of blood cells yellowish, the nucleus black, and the elastic fibres of the vascular walls are stained dark red.

Assessment of the articular cartilage included examination of its surface smoothness, thickness of the layer, number of cells, their size and distribution in the depth dimension, cell agglomerations and nucleation of the cells. Separate attention was paid to the subarticular spongy bone and to the nucleation of the osteocytes situated at greater depth, to the occurrence of osteoblasts and osteoclasts, and in the bone marrow to vascularization and content of cells, and above all to the presence of granulation tissue and its place and extent of occurrence. The ossification of the epiphyseal cartilage was studied in the operated and unoperated extremity in respect of ossification, column width, arrangement of cells and nucleation of the cells.

The histochemical methods employed in this work for investigation of the ground substance in the connective tissue were the Alcian blue stain and the periodic acid Schiff (P.A.S.) oxidation method.

Alcian blue is a selective stain for acid polysaccharides devised by STEEDMAN (1950). LISON (1954) reports on the usefulness of this new method, stating that it is also particularly well suited for investigation of cartilage and especially of ossification. It seems according to him that stainability with Alcian blue is proportional to the chondroitin sulphuric acid content of the cartilage. It imparts a bluish green stain to the ground substance of cartilage and to certain types of tissue, purplish blue to the nucleus, red to collagen and bone, and a pale yellow to cytoplasm and muscle. In the present work LISON's technique was employed in which Chlorantine Fast Red 5 B is added to the basic stain.

The P.A.S. staining was done according to McMANUS (1948). This is a histochemical reaction in the first stage of which the tissue polysaccharides are converted to polyaldehydes under periodic acid treatment. In the second stage these polyaldehydes are made visible by fuchsin sulphuric

acid, which is a carbonyl reagent. Cartilage responds with the heaviest violet stain because cells and nuclei are both stained. In the bone matrix an intensive stain is clearly visible upon decalcification. The stain demonstrates the general presence of carbohydrates without being more specific.

Autoradiographic examinations were carried out in eight cases. An injection of 3.0 millicuries of  $P^{32}$  in the form of phosphate (from the Radiochemical Centre, Amersham, England) was given intravenously to the anaesthetized animals two hours before they were sacrificed. After splitting of the femoral head through the middle in the frontal plane with an electric saw, one of the halves was fixed in 75 per cent alcohol and treated by the methyl metacrylate procedure without decalcification. The specimens were ground down to 100  $\mu$  thickness. Autoradiographs were then prepared according to the contact method on Kodak Fast Autoradiographic Stripping Plates AR 50. The contact plates were exposed and developed in Kodak D19-80 Developer. The time of exposure, six days, had been determined in preliminary tests.

In order to obtain a histologic reference series for those cases in which autoradiography was undertaken, the other split half of the femoral head was decalcified as usual and fixed and stained with Weigert van Gieson's haematoxylin.

Micro-radiography is a procedure by which an absorption roentgenograph is obtained with x rays in 1:1 scale of the specimen under investigation. The roentgenogram is then examined under the microscope. Micro-radiographs were here prepared of bone specimens ground down to 100  $\mu$  thickness. Because the specimens contain biologic material, this investigation is referred to as historadiography (ENGSTROM 1955). The present historadiographs were made with a Philips X-ray Apparatus Type 11704 using x-ray tube No. 25293 32. The specimen was placed 10 cm from the copper anode and the film was exposed for 2 minutes at 20 kV and 15 mA. The sensitive material was Kodak's Spectroscopic Plate 649-GH which was developed during 5 minutes (experimentally determined) in Kodak D 19 b developer.

The different studies that were made are tabulated in Table 1.

The results of the macroscopic, roentgenologic and microscopic examination are presented in connection with each group of experimental animals in Tables 2—7 and according to method of examination in Tables 8—10 in the discussion. This duplication was thought to be helpful to the reader.

## ARTHROTOMY DIVISION OF LIGAMENTUM TERES AND TEMPORARY LUXATION OF THE FEMORAL HEAD

### EARLIER INVESTIGATIONS

*Clinical investigations* — To a great extent the investigations have reference to the blood vessels passing through the ligamentum teres and to their significance in various age periods and in various situations KOLODNY (1925) found that this ligament was significant only in children and that it goes to the centre of ossification while NUSSBAUM (1924-1926) claimed the direct opposite as did also WALMSLEY (1915) and WOLCOTT (1933)

Numerous subsequent investigations show that in children as well as in adults the vessels of lig. teres are in function in most instances (CHANDLER and KRELSCHER 1932 SCHWEIGERT 1936 NORDENSON 1938 VEREBY 1942 TICKNER 1949 HOWE 1950 CHEYNEL 1954 and JUDIT *et al* 1955) Some investigators (LANGER 1875 WALMSLEY 1915 LANG 1916 KOLODNY 1925 CHEYNEL 1947 and ETIENNE and GRANEL 1949) arrived at the result that with age the blood vessels decrease in number but this has been contradicted by others (HEINE 1926 and BENNETT *et al* 1942)

WALDENSTROM (1934) stated that in the treatment of epiphysiolysis of the femoral head either operatively or by reposition necrosis of the femoral head is due to damage of the lig. teres

*Animal experiments* — ZEMANSKY and LIPPMAN (1929) divided the lig. teres and part of the capsule of two-weeks old rabbits and observed after 6-35 days levelling of the femoral head and avascular changes but no regeneration In three rabbits of seven weeks age which were operated to serve as controls no corresponding changes could be found GRAHAM (1930) divided the lig. teres of young goats producing flattening of the femoral head and partial necrosis STEWART (1933) divided the lig. teres of young and adult rabbits and of adult dogs He sacrificed the animals after 7-120 days and observed slight changes of bone in the young as well as old animals By division of the lig. teres in cats dogs and rabbits of various ages CELLA (1934) obtained changes in the femoral head in those aged 1-10 days while no changes developed in animals 40 days to 5 months

acid which is a carbonyl reagent. Cartilage responds with the heaviest violet stain, because cells and nuclei are both stained. In the bone matrix an intensive stain is clearly visible upon decalcification. The stain demonstrates the general presence of carbohydrates, without being more specific.

Autoradiographic examinations were carried out in eight cases. An injection of 3.0 millicuries of  $P^{32}$  in the form of phosphate (from the Radiochemical Centre, Amersham, England) was given intravenously to the anaesthetized animals two hours before they were sacrificed. After splitting of the femoral head through the middle in the frontal plane with an electric saw, one of the halves was fixed in 75 per cent alcohol and treated by the methyl metacrylate procedure without decalcification. The specimens were ground down to 100  $\mu$  thickness. Autoradiographs were then prepared according to the contact method on Kodak Fast Autoradiographic Stripping Plates AR 50. The contact plates were exposed and developed in Kodak D19 Developer. The time of exposure, six days, had been determined in preliminary tests.

In order to obtain a histologic reference series for those cases in which autoradiography was undertaken, the other split half of the femoral head was decalcified as usual and fixed and stained with Weigert van Gieson's haematoxylin.

Micro-radiography is a procedure by which an absorption roentgenograph is obtained with x rays in 1:1 scale of the specimen under investigation. The roentgenogram is then examined under the microscope. Micro-radiographs were here prepared of bone specimens ground down to 100  $\mu$  thickness. Because the specimens contain biologic material, this investigation is referred to as *historadiography* (ENGSTROM 1955). The present *historadiographs* were made with a Philips X-ray Apparatus Type 11704 using x-ray tube No. 25293/32. The specimen was placed 10 cm from the copper anode and the film was exposed for 2 minutes at 20 kV and 15 mA. The sensitive material was Kodak's Spectroscopic Plate 649 GH which was developed during 5 minutes (experimentally determined) in Kodak D19b developer.

The different studies that were made are tabulated in Table 1.

The results of the macroscopic, roentgenologic and microscopic examination are presented in connection with each group of experimental animals in Tables 2—7 and according to method of examination in Tables 8—10 in the discussion. This duplication was thought to be helpful to the reader.



the joint was fully comparable to that of the intact side the sole difference consisting of insignificant scar formations. At 40 days, but not in any other specimen unevenness was noted in the articular cartilage. Owing to the small size of the hip joint of the rabbit it is not possible actually to speak of the quantity of articular fluid, at its most one may distinguish a moist condition of the joint. Thus macroscopic comparisons in this respect appeared too inaccurate. The lig. teres had become reattached in four instances *ie.*, at 10, 25, 100 and 110 days.

*Roentgenologic changes* — As a rule no changes were visible. In contrast with this however 100 days after the operation there could be seen, in addition to subluxation an increased density of the head of the left femur greater translucency close to the articular cartilage and sclerosis at the upper margin of the acetabulum (Fig. 3).

At 140 days similar changes were observed the femoral head showed increased density the sclerosis in the upper part of the acetabulum was



Fig. 3. X-ray 100 days after arthrotomy division of lig. teres and temporary luxation of the femoral head. In addition to subluxation there is increased density of the head of the left femur greater radiolucency close to the articular cartilage and sclerosis at the upper margin of the acetabulum.



accentuated and the entire acetabulum presented a confused structure. A distinct spot of greater translucency was observed in the distal part of the femoral head. At 180 days after the operation the joint space appeared to be narrower on the left side (Fig. 4).

*Microscopic changes — 5 — 15 days* In the specimen of the 5th day the articular cartilage was thin, but its surface was smooth. In that of the 10th day the articular surface was uneven and frayed in spots. The specimen taken on the 15th day presented a fairly smooth articular cartilage but it had been destroyed and was replaced by fibrous tissue in the peripheral part near the epiphysis (Fig. 5). A somewhat similar change was also seen in the periphery on the other side. At the corresponding points of the femoral head osteoblasts were seen in the spongy bone adjacent to the destroyed articular cartilage.

*15 — 45 days* In the specimen of the 25th day the articular surface was uneven and frayed in spots. There were no bone changes. In the specimen

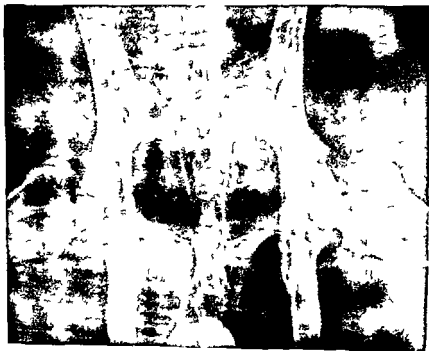
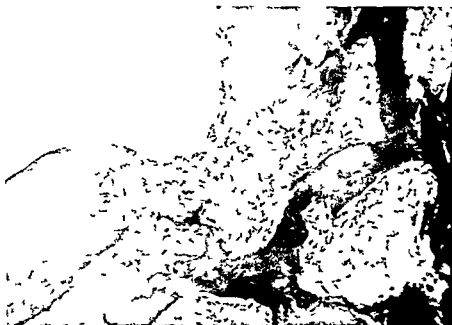


Fig. 4 X ray 180 days after arthrotomy division of lig. teres and temporary luxation of the femoral head. The joint space appears to be narrower on the left side.



*Fig 5 Micrograph 15 days after arthrotomy division of lig teres and temporary luxation of the femoral head In the peripheral part of the articular cartilage on the side of the epiphyseal cartilage the articular cartilage seems to be destroyed and replaced with fibrous tissue — Alcian blue  $\times 100$*



*Fig 6 Micrograph 30 days after arthrotomy division of lig teres and temporary luxation of the femoral head The articular cartilage is normal in thickness on one margin — Weierstrass-Gieson  $\times 100$*

taken on the 30th day granulation tissue was present in the cancellous spaces under the ragged articular cartilage but there was no osteogenesis. In the specimen of the 35th day the articular cartilage was of normal thickness on one margin and replaced by fibrous tissue on the other. Also elsewhere it was uneven and frayed (Figs 6 and 7). No bone changes were noted however. In the specimen of the 40th day the articular cartilage had vanished over a large portion and was replaced by fibrous tissue and fibrous cartilage. In the spongy bone Alcian blue revealed lighter areas in which no nuclei were visible while the nucleated areas stained a brighter red.

45—90 days. In the specimen of the 80th day the articular cartilage was uneven and frayed here and there. At 90 days poorer staining of the nuclei was observed over an extensive area on the inner side of the articular cartilage and the cells in the bone marrow were few in number.

90—240 days. In the specimen of the 100th day the articular surface was here and there uneven and frayed. The bone presented a few areas poor in nuclei. The cancellous spaces were normal elsewhere but there was granulation tissue in two or three spaces near the articular cartilage. In the specimens taken at 110, 120 and 130 days some slight unevenness of the articular cartilage was observed, but nothing abnormal in the bone. In the specimen of the 140th day the articular cartilage was uneven and frayed in spots and it was disrupted at one point in the periphery. Beneath it there was granulation tissue and areas poor in nuclei were found in the bone itself in addition to which occasional osteoclasts and osteoblasts were seen (Figs 8 and 9).

In the specimen taken 150 days after the operation the articular cartilage was replaced at one point by tissue resembling fibrous and hyaline cartilage (Fig. 10).

In the specimen of the 180th day the articular surface was uneven and frayed in spots but no bone changes were observed. The specimen of the 240th day presented an articular cartilage of partly faultless surface but lightly frayed at one margin in addition lacunae were noted each containing several cartilage cells although these cells usually occurred singly in pairs or in threes in the areas of normal cartilage. There were no bone changes.

Of the above mentioned specimens those taken at 10, 25, 100 and 110 days exhibited reattachment of the ligaments.

Epiphyseal cartilage was observed in seven instances among the specimens taken 5—50 days after the operation and only once subsequently, namely in the specimen of the 60th day. In the specimen of the 5th day



Fig 7 Micrograph 35 days after arthrotoomy division of ligaments and temporary luxation of the femoral head of the other margin of the same specimen as in Fig 6 showing articular cartilage of frayed structure poor in cells and less intensively staining — Weierstrass-Gieson  $\times 100$



Fig 8 Micrograph 140 days after arthrotoomy division of ligaments and temporary luxation of the femoral head. The articular cartilage has subsided and the bone under it is destroyed but partly replaced by granulation tissue. The articular surface is uneven — Weierstrass-Gieson  $\times 100$

Fig 9 Detail of the same micrograph as in Fig 8 showing bone in the process of destruction by side it osteoclasts and more in the center incipient formation of new bone osteoblasts and granulation tissue — Weigert van Gieson  $\times 500$

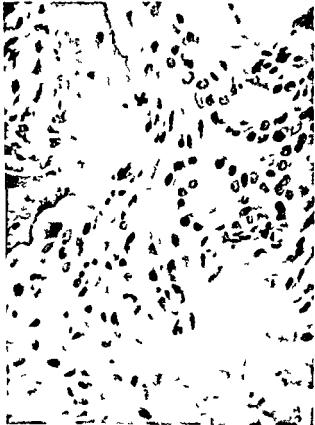


Fig 10 Micrograph 120 days after arthrotomy division of ligaments and temporary fixation of the femoral head. The articular cartilage has been destroyed at one point and replaced by cartilage resembling fibrous and hyaline cartilage — Weigert van Gieson  $\times 100$



ossification of the epiphysis was found to be farther advanced in the operated hip

Necrosis of the bone was found in 4 instances among the 22 operated rabbits

#### COMMENTS

In earlier investigations distinct changes were induced in the femoral head and articular cartilage of very young rabbits and other experimental animals already by the performance of arthrotomy and division of lig teres while little or no changes resulted in older animals. In the present investigation unevenness of the surface of the articular cartilage was found microscopically in 15 instances. In the specimen taken 15 days after operation the degenerative changes in the articular cartilage were marked and in the same specimen there was also granulation tissue in the subcartilaginous bone. According to the literature a much longer time is required for degeneration and necrosis of cartilage and its replacement to occur (HALDEMAN 1938 CARLSON 1957). One might therefore suspect that in this case the change had been present already prior to operation. However no equivalent changes were visible in the control specimens.

The other changes observed in the cartilage may in part have been outright artefacts produced by instrumental interference or they may have been caused traumatically by a slight change in position of the femoral head due to the arthrotomy and division of lig teres itself. Rather exceptionally in four instances necrotic changes occurred in the bone. They were concomitant in three cases with degenerative changes in the cartilage. It would thus seem that the bone reacts rapidly to changes in the cartilage possibly with the aim of strengthening the static condition of the femoral head at such a point or of enhancing the regeneration of cartilage by this means.

However no alterations were observed in the articular cartilage at 90 days although in an extensive area of the bone adjacent to the articular cartilage poorer staining of the nuclei and scantiness of cells in the bone marrow were noted. In this case it may have been a question of degeneration of bone on a circulatory basis encountered in connection with the normal aging of bone which has previously been demonstrated by e.g. MULLER (1927) JAFFE and POMERANZ (1934) and SHERMAN and SELAKOVICH (1957). It is a fact, at least that no subchondral fracture as described by NAGLRA (1937 1938) was observed which would have accounted for such a change. On the other hand the change was of such extent that one would expect some other factor, for instance operative

trauma of exceptional magnitude to have accelerated in this case the process that led to necrosis of the bone

After reattachment of the lig. teres the specimens differed in no way from other virtually simultaneous specimens

In two instances increased roentgenologic density was encountered which was then correlated with the necrotic changes found in the bone. However a comparatively long time had passed since the operation in both cases and it is therefore not possible to speak of a roentgenologic early diagnosis

Summarizing what has been stated above it can be said that the macroscopically observed changes in the femoral head were fairly insignificant. Increased roentgenologic density was noted twice at the femoral head and in these cases granulation tissue was histologically observable in the cancellous spaces in addition to which there were areas poor in nuclei in the femoral head itself. Numerous specimens revealed microscopically unevenness of the articular cartilage and its slight destruction and replacement with fibrous cartilage or fibrous tissue with granulation tissue reaction in the corresponding underlying area. An actual change indicative of partial necrosis of the bone was found in four instances. In these cases the areas poor in nuclei took a lighter red stain with Alcian blue.

*On the strength of the investigations carried out in the present connection my opinion is that the observed changes in the articular cartilage had mainly occurred as a consequence of direct trauma. Furthermore they were insignificant in magnitude since they were rarely accompanied by granulation tissue reaction. Some correlation with simultaneous changes in the bone was noted.*

ossification of the epiphysis was found to be farther advanced in the operated hip

Necrosis of the bone was found in 4 instances among the 22 operated rabbits

#### COMMENTS

In earlier investigations distinct changes were induced in the femoral head and articular cartilage of very young rabbits and other experimental animals already by the performance of arthrotomy and division of lig teres while little or no changes resulted in older animals. In the present investigation unevenness of the surface of the articular cartilage was found microscopically in 15 instances. In the specimen taken 15 days after operation the degenerative changes in the articular cartilage were marked, and in the same specimen there was also granulation tissue in the subcartilaginous bone. According to the literature a much longer time is required for degeneration and necrosis of cartilage and its replacement to occur (HALDEMAN 1938 CARLSON 1957). One might therefore suspect that in this case the change had been present already prior to operation. However no equivalent changes were visible in the control specimens.

The other changes observed in the cartilage may in part have been outright artefacts produced by instrumental interference or they may have been caused traumatically by a slight change in position of the femoral head due to the arthrotomy and division of lig teres itself. Rather exceptionally in four instances necrotic changes occurred in the bone. They were concomitant in three cases with degenerative changes in the cartilage. It would thus seem that the bone reacts rapidly to changes in the cartilage possibly with the aim of strengthening the static condition of the femoral head at such a point or of enhancing the regeneration of cartilage by this means.

However no alterations were observed in the articular cartilage at 90 days although in an extensive area of the bone adjacent to the articular cartilage poorer staining of the nuclei and scantiness of cells in the bone marrow were noted. In this case it may have been a question of degeneration of bone on a circulatory basis, encountered in connection with the normal aging of bone which has previously been demonstrated by e.g. MULLER (1927) JAFFE and POMERANZ (1934) and SHERMAN and SELAKOVICH (1957). It is a fact at least, that no subchondral fracture as described by NAGURA (1937 1938) was observed which would have accounted for such a change. On the other hand the change was of such extent that one would expect some other factor for instance operative



cent TROJAN and PERLSCH (1956) 2.9 per cent in common fractures and 19.4 per cent in fractures with associated luxation BANCROFT and MURRAY (1945) degenerative changes ensuing in 50 per cent and DUBIGNEF (1956) 64 per cent WALLER (1955) noted that in dashboard fractures avascular bone necrosis occurred in 16.6 per cent and traumatic arthritis in 44.8 per cent These figures refer to varying lengths of observation period and to different degrees of severity of the original trauma but they serve to indicate the general trend and they bear evidence of the importance of bone necrosis

*Animal experiments* — MULLER (1924) who investigated traumatic luxation of the hip joint in animals of various ages and who was the first to clarify the histologic features encountered in this connection observed that luxation of the joint caused necrosis of cartilage only in adolescent animals during an observation period of 6—40 days whereas in adult animals he found fibrous tissue in the cancellous spaces and areas poor in cells in the bone.

BERGMAN (1927) produced closed luxation in two adolescent rabbits observing in one of them which was sacrificed after two months that part of the bone trabeculae under the articular cartilage stained poorly and that the articular cartilage was interrupted in many places LANGENSKIÖLD and SARPIO (1958) luxated the hip joint in rabbits a few days old observing after a few weeks in addition to dysplasia of the acetabulum hypoplasia of the epiphysis of the femoral head SMITH *et al* (1958) produced operative luxation of the hip joint in 16 young dogs aged three to five weeks obtaining considerable coxa vara and reduced anteversion They sacrificed the animals after closure of the epiphysis and no histologic examination was carried out

In their investigation of the influence exerted on the articular cartilage by functional activity of the joint in rabbits EKHOLM and NORBACK (1951) observed that already after a short time of activity the cartilage cells were more rounded in the surface layer owing to a greater quantity of fluid INGELMARK (1950) studied the value of the joint fluid in the supply of nutrition and inclined towards the opinion that the principal nutrition comes by way of the bone and not from the articular fluid This had also been indicated earlier by the experiments of INGELMARK and SÄF (1946) On the other hand BROWER *et al* (1962) observed in their experiments with rabbits that 0.5 per cent neutral red solution entered the cartilage by way of the articular fluid KETTUNEN (1958) noted in his studies of skin arthroplasty in cats the significance of function with regard to the metaplasia of fibrous tissue into cartilaginous tissue



## OWN INVESTIGATIONS

Rather few animal experiments have been carried out on this subject and the following study therefore seemed called for

The macroscopic roentgenologic and microscopic findings in the experiments with arthrotomy and luxation without reduction (Group II) are shown in Table 3

*Macroscopic changes* — Unevenness of the surface of the articular cartilage was observed in nearly all specimens. The changes were distinct already 20 days after the operation the articular cartilage had lost its normal gloss a new capsular sheath had been formed of the surrounding tissue and the femoral head had begun to grind a new socket for itself in the pelvic bones dorsal and cranial to the original acetabulum. At later times the changes were more massive and the femoral head had also lost its normal shape and hardness. The femoral head had returned into its original socket in two instances namely in the specimens taken at 40 and 70 days. In these cases the changes in the surroundings were rather insignificant.

*Roentgenologic changes* — Twice (in the animals sacrificed at 40 and 70 days) the femoral head was seated in its proper socket. At 30 35 40 days (Fig. 11) 50 60, 80, 90 100 110 days (Fig. 12) 130 140 150 180 and 240 days after the surgical interference increased density was noted in all the femoral heads and small areas of greater radiolucency now and then in the femoral head but also in the neck of the femur. In most cases there also were observed a distinctly altered shape of the femoral head and degenerative changes in the pelvic bones. It was found that the femoral head had hollowed for itself a new socket cranial and dorsal to the initial acetabulum. In these cases the joint space displayed slightly greater width in nearly all  $\lambda$  rays.

*Microscopic changes* — 5—15 days. At 5 days the articular cartilage was found to be uneven and frayed but there were no bone changes. In the specimen taken at 10 days were noted unevenness of the articular cartilage and at one point fibrous tissue on the articular surface and some granulation tissue in the cancellous spaces at the margin of the femoral head (Fig. 13). In the specimen of the 15th day no changes were seen in the articular cartilage but there was some granulation tissue in the cancellous spaces and slight occasional scantiness of cells and disappearance of nuclei in the spongy bone itself.

15—40 days. On the 20th day the articular cartilage was found to be uneven and frayed and there was granulation tissue in the cancellous



*Fig 11 X ray 45 days after arthrotomy and luxation of the femoral head without reduction The luxated femoral head has ground a new socket for itself cranial to the original acetabulum The femoral head appears to be of considerably greater density on the operated side*



*Fig 12 X ray 110 days after arthrotomy and luxation of the femoral head without reduction Extensive degenerative changes not only in the femoral head but also in the pelvic bones and the trochanteric region on the left side increased density as well as areas of greater radiolucency in spots*



Fig 13 Micrograph 10 days after arthrotomy and luxation of the femoral head without reduction. At one point the articular cartilage has been deeply lacerated. — Allan Blair 100



Fig 14 Micrograph 100 days after arthrotomy and fixation of the femoral head without reduction. The articular cartilage has almost entirely been replaced by fibrous cartilage and fibrous tissue. In the underlying anellous spaces there is abundant fibrous tissue and osteogenesis. — Wegerstein Giesse 40



*Fig 15 Micrograph 100 days after arthrotomy and luxation of the femoral head without reduction. Large cancellous spaces contain abundant granulation tissue and osteogenesis — Alcian blue  $\times 280$*



*Fig 16 Micrograph 240 days after arthrotomy and luxation of the femoral head without reduction. Abundant granulation tissue in the cancellous spaces and osteogenesis — Alcian blue  $\times 280$*

spaces In the specimen of the 30th day the articular cartilage was worn down and thin it was completely absent in spots and granulation tissue was observed in the cancellous spaces Examination of the specimen of the 35th day revealed in addition to the foregoing areas of articular cartilage and granulation tissue in the cancellous spaces The spongy bone was poor in cells and osteoblasts and foamy degeneration of fat cells were encountered in the cancellous spaces in the middle of the specimen In the specimen of the 40th day the articular cartilage was very slightly uneven and a couple of smaller particles had become detached from it but no bone changes were observed At 45 days the articular cartilage was found to be uneven and frayed and the cancellous spaces contained granulation tissue

45—90 days In the specimen taken at 50 days most of the articular cartilage had disappeared and was replaced by fibrous tissue Granulation tissue was seen in the cancellous spaces under the articular cartilage on one side The specimen of the 60th day had an articular cartilage that was even in some places but it was absent and replaced by fibrous tissue in others the spongy bone under the articular cartilage was poorer in cells and there was granulation tissue in the cancellous spaces In the specimen of the 70th day the articular cartilage was mostly destroyed and replaced either by fibrous tissue or in spots, by cartilage with hyaline content Granulation tissue was highly abundant in the cancellous spaces while the spongy bone itself was sparse in quantity and contained areas poor in cells or displaying lack of nuclei The articular cartilage in the specimen of the 80th day was very narrow and frayed and cell agglomerations were observable in it The articular cartilage was not compressed The femoral head on the other hand was found to be flattened throughout very abundant granulation tissue and protuberant osteogenesis osteoclasts and osteoblasts were seen in the cancellous spaces The spongy bone contained extensive areas without nuclei and numerous areas poor in nuclei In the specimen of the 90th day the articular cartilage had disappeared almost completely and was replaced by fibrous cartilage and fibrous tissue Large cancellous spaces rich in granulation tissue and extensive areas of anuclear bone tissue were also noted Osteoclasts and osteoblasts were abundant The areas poor in nuclei stained a lighter red with Alcian blue while new bone was a brighter red In the P A S stained slides the areas where no nuclei were seen were a lighter violet in colour

90—240 days Changes of extraordinary degree were observed in the specimens referring to the period of 100—180 days Only fragments were left of the articular tissue embedded in fibrous tissue and at numer

ous points the articular cartilage had been replaced by tissue resembling hyaline cartilage or by fibrous cartilage. The changes in the cancellous spaces and in the spongy bone in the specimens taken at 100 days (Figs 14 and 15) and at 110, 120, 130, 140 and 150 days were similar in kind. In the specimen of the 120th day furthermore the femoral head appeared flattened. In the specimen of the 180th day granulation tissue was observed in particular abundance and osteoclasts and osteoblasts in profuse quantity in the entire femoral head. In the specimen of the 240th day some larger portions of the articular cartilage were still comparatively well preserved but they were covered with fibrous cartilage. The greater part of the articular cartilage however, was destroyed and fibrous tissue and fibrous cartilage occupied its place. The changes in the cancellous spaces and in the spongy bone were like those in the preceding specimens (Fig 16). In the preparations stained with Alcian blue the new bone was bright red but also less intensively stained anuclear areas were seen.

Epiphyseal cartilage was observed in six instances among the specimens referring to periods of observation between 5 and 50 days and twice thereafter namely in the specimens of the 110th and 120th days. In the specimens of the 35th, 40th and 45th days ossification of the epiphyseal



*Fig 17 Contact autoradiographs 90 days after arthrotomy and fixation of the femoral head without reduction. Uniform uptake of radio-phosphorus on the intact right side (control) and uneven distribution on the operated side with greater deposition in the area of osteogenesis and scantier deposition where necrosis of the bone was observed.*



line was found to be farther advanced in the operated hip whereas it was less strongly ossified in the specimen taken on the 5th day.

*Autoradiographic changes* — Two specimens namely, those of the animals sacrificed 90 and 240 days after the operation were subjected to autoradiographic study. Comparison of the specimens from both hips in the first mentioned case revealed (Fig. 17) that the structure was distinctly reproduced in the autoradiograph of the control side (right hip) where the radioactive phosphorus ( $P^{32}$ ) was uniformly taken up by the bone tissue while the deposition on the operated side was less uniform. More radio phosphorus was observed in the area where osteogenesis occurred.



Fig. 18. Autoradiograph 90 days after a throtomy and luxation of the femoral head without reduction. Distinct differences observable between granulation tissue (1) tissue in the process of osteogenesis (2) and normal bone (3). Likewise necrotic bone (4) displays stronger x-ray absorption producing a lighter area in the radiograph.

ous points the articular cartilage had been replaced by tissue resembling hyaline cartilage or by fibrous cartilage. The changes in the cancellous spaces and in the spongy bone in the specimens taken at 100 days (Figs 14 and 15) and at 110, 120, 130, 140 and 150 days were similar in kind. In the specimen of the 120th day furthermore the femoral head appeared flattened. In the specimen of the 180th day granulation tissue was observed in particular abundance and osteoclasts and osteoblasts in protuberant quantity in the entire femoral head. In the specimen of the 240th day some larger portions of the articular cartilage were still comparatively well preserved but they were covered with fibrous cartilage. The greater part of the articular cartilage however was destroyed and fibrous tissue and fibrous cartilage occupied its place. The changes in the cancellous spaces and in the spongy bone were like those in the preceding specimens (Fig. 16). In the preparations stained with Alcan blue the new bone was bright red but also less intensively stained anuclear areas were seen.

Epiphyseal cartilage was observed in six instances among the specimens referring to periods of observation between 5 and 50 days and twice thereafter namely in the specimens of the 110th and 120th days. In the specimens of the 35th, 40th and 45th days ossification of the epiphyseal

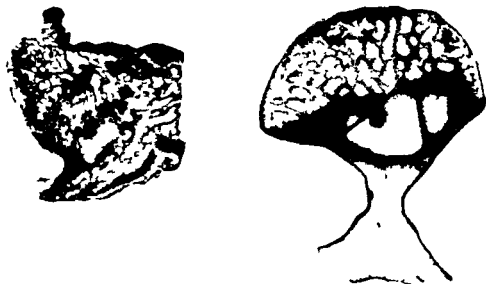


Fig. 1. Contact aneurysm, 35 days after arthrotomy and luxation of the femoral head without reduction. Uniform deposits of radio-phosphorus on the smooth articular side (control) and uneven distribution on the perched side in the greater deposit on the area of asperities and earlier deposition where necrosis of the bone was observed.

factors than bone but on the other hand its repair was slower. It should be noted however that the articular cartilage was in a less favourable position in the present experiments than the bone. The effect of pressure, the altered statics and the direct trauma all acted directly on the articular cartilage and less on the bone protected by the cartilage and supplied with nutrition by the cancellous circulation.

According to EKHOLM and NORBACK (1951) the function of the joint is an essential factor in the aetiology of changes occurring in the cartilage. Lack of function in itself may cause necrosis of the cartilage. KULTUNEN (1958) observed in his experimental investigation with skin arthroplasty in cats that metaplasia of cartilage took place in the grafted connective tissue only under the influence of function. As in the present work the extremities had not been immobilized in any manner there can be no question of lack of function at least in the initial stage. On the contrary, seeing that the unprotected articular cartilage had been forced to grind a new socket the contribution of trauma was obviously considerable indeed. The nutrient effect of the articular fluid was insignificant. It has in fact been suggested that the articular cartilage receives its principal nutrition from the bone (INGELMARK and SAAF 1946; INGELMARK 1950) though there also are reports that it receives its nutrition by mediation of the articular fluid according to the investigations of BROWER *et al* (1962). The comminution of the articular cartilage causes a regenerative reaction in the underlying bone with increasing granulation tissue as could be seen in the course of 60 days.

After 70 days the extensive changes in the articular cartilage were supplemented not only by an increase of granulation tissue but also by the occurrence of osteoblasts, osteoclasts and necrosis of bone particularly in the middle portion of the femoral head. Obviously the pressure, the changed statics and the reduced function also exerted their influence on the bone so that the spongy bone which had become superfluous disappeared and was replaced by new bone tissue that was formed at a more expedient point.

According to HARRISON *et al* (1953) hyperaemia plays an important part in the aetiology of osteoarthritis. On the other hand RUTISHAUSER (1956) and HULTIN (1958) claimed that osteoarthritis may develop on the basis of venous stasis. It is obvious that pressure, changed statics, direct trauma and reduced function are essential agents in producing necrosis of cartilage and by reflectory response necrosis of bone. These factors are also present in osteoarthritis. BOHLER (1957) also observed in his experimental investigation of luxation the essential significance of direct

specifically of acute trauma as a primary circumstance in the aetiology of necrosis of the bone

It was found that the onset of cartilaginous changes occurred rapidly after luxation. In the two cases in which the femoral head had returned again into its socket the changes in the specimen taken at 40 days were very few and the reposition had obviously occurred within a rather short time after the luxation whereas in the specimen of the 70th day there were copious changes and the luxated condition had apparently persisted for a long time in this instance

The autoradiographic studies with  $P^{32}$  revealed that bone in necrosis took up less phosphorus than living bone. On the other hand more phosphorus was deposited in newly formed bone. In the x rays as was also the case in microradiography the changes were also of a kind associated with the reaction of bone necrosis so that after a longer period the necrosis of bone itself caused distinctly increased density

Summarizing what has been stated above it can be said that abundant macroscopic changes were observed in the femoral head and in its articular cartilage also with the exception of one single case there was a new socket and in two instances spontaneous reposition of the femoral head in its socket. Roentgenologically too the changes were quite distinct. In addition to changes in the joint space and those relating to displacement of the femoral head there were roentgenologically distinct degenerative changes in the femoral head itself in cases in which the histologic picture displayed necrosis in the bone in addition to strong granulation tissue reaction. However fibrous tissue reaction in the cancellous spaces alone did not produce any roentgenologically observable changes as could be noted in four instances. Among the microscopic findings degenerative changes in the articular cartilage were dominant initially and the cartilage had frequently disappeared almost entirely. In addition to changes of the articular cartilage granulation tissue reaction was observed in the cancellous spaces and excepting only two cases actual necrosis of bone was not encountered until beginning on the 60th day the histologic picture being governed even then by extraordinarily strong granulation tissue reaction and abundance of osteoclasts and osteoblasts

Necrosis of the bone was found in altogether 11 instances. The autoradiographic changes showed up a clear difference between the two sides in respect of regularity of structure the bone structure of the intact control side being clearly visualized. On the operated side powerful deposition of radio-phosphorus in the area of osteogenesis was noted

in contrast to poor uptake at the point where necrosis occurred. In the microradiographs the mass of the necrotic bone tissue was greater than that of the surrounding bone tissue.

*It is my opinion that the observed profuse changes in the articular cartilage with the associated granulation tissue reaction are due to direct trauma. Later the pressure changed static conditions and reduced function also affect the bone in which in addition to degenerative changes abundant regeneration is observed.*

## ARTHROTOMY AND CAPSULOTOMY

### EARLIER INVESTIGATIONS

*Clinical and experimental investigations* — It has already been pointed out in the foregoing that luxation of the femur frequently causes aseptic necrosis of bone. Arthroplasty, of whatever kind it may be, always necessitates luxation of the joint. In describing, in CAMMILLI'S textbook of operative orthopaedics (1956) different methods of opening the hip joint, KNIGHT only once mentioned possible vascular lesion in this connection, namely, in his account of Ollier's lateral U incision where he cautioned against damaging the capsular branch of the medial circumflex artery. In numerous other connections only the direction of the capsular incision is mentioned. For instance MARCY and FLETCHER (1954) and LICK (1955) said nothing about the capsular vessels whereas e.g. McIVARLAND and OSBORN (1954) reminded of the branch of the medial circumflex artery behind the trochanter and of that of the lateral circumflex artery in the neighbourhood of the vastus lateralis.

PHILMISTIR (1930) described a case of necrosis in the femoral head five months after arthroplasty performed with the aid of the fascia lata in a man aged 28. SMITH PETERS (1949) in describing the incision employed by him in his cup arthroplasty, warned against causing distal damage to the capsule. JAKOBSEN (1957) stated in his investigation concerning the same operation that vessels may be damaged in any of the following ways: by injury to small vessels where they penetrate the bone at the osteophyte ring when the femoral head is being scraped; by excessive radical resection of the lateral part of the articular capsule whence the main blood supply comes; or by detaching the greater trochanter and thereby destroying the greater part of the vessels supplying the trochanter and the neck of the femur. Since atrophy has rarely been encountered in osteoarthritis, this has to be considered a postoperative complication resulting from the above described vascular lesion. JAKOBSEN stated that there was roent-

genologically atrophy in 16 per cent of cases postoperatively and that in one such reoperated case total necrosis of the femoral head and of the neck of the femur was seen

JUDET *et al* (1955) studied in cadavers the blood supply of the femoral head and neck by Trueta's method. At first they performed on eight bodies the arthroplastic operation devised by themselves. Subsequently, on examination of the blood supply, they observed that particularly drilling of the centre and shaping of the stump produced serious disturbances of circulation within the bone and in the medial inferior arteries respectively. Examination of the lesions produced in connection with opening of the hip joint in 15 cadavers revealed that no disturbances of metaphyseal or epiphyseal vascularization are caused even by T capsulectomy. If the point of attachment of the capsule to the bone is not disturbed, not even complete anterior or posterior capsulectomy will cause reduction of the blood supply to the neck of the femur, but it will reduce the blood supply to the femoral head. If the attachment of the capsule is destroyed, disturbances follow also in the epiphysis. The authors studied Hueter's anterior approach in seven cadavers and Gibson's posterolateral incision in eight, finding that they carried no significance in respect of vascular lesions unless excessively distal capsulectomy was performed or the arteries on the upper surface of the neck of the femur were damaged.

Also the medial and lateral circumflex arteries could be ligated without incurring any noteworthy changes. Likewise the point of origin of the deep femoral artery and of the circumflex arteries could be ligated without causing marked reduction of the blood flow in the femoral head or neck. Neither was any change caused by ligation of the circumflex arteries and the quadriceps artery and superficial femoral artery 10 cm below the point of origin of the deep femoral artery, or on the other hand by ligation of the deep femoral artery below the origin of the circumflex artery and simultaneous ligation of the superficial femoral artery 2 cm below the origin of the deep femoral artery.

LAURENT (1953) stated in connection with discussion of the operative treatment of congenital dislocation of the hip in children, that the capsule must not be divided at the point of lateral insertion, as haemorrhage will occur from the capsular branch of the femoral circumflex artery if the capsule is opened in its ventral distal part. In a later report (LAURENT 1959) he pointed out that in conservative as well as operative orthopaedic treatment of congenital dislocation of the hip care should be taken not to cause epiphyseal damage, which is the major reason for poor results. PYLKKÄNEN (1960) stressed the importance of avoiding damage to the

capsular vessels in connection with operations of the hip particularly in children

*Animal experiments* — KISTLER (1934) ligated the lig. teres and caused injury to the vessels in the trochanteric region in rabbits aged 3 weeks to 2 years. On sacrificing the animals after periods of 1—150 days he found changes simulating osteochondritis juvenilis in young animals, but in the older rabbits lesion of the lig. teres alone produced no such changes unless the vascularization of the trochanteric region was simultaneously damaged. No other references to investigations with a bearing on total capsulectomy have been found in the literature.

#### OWN INVESTIGATIONS

Although the significance of the capsule and its preservation have been commented upon in considerable extent in the literature relatively little experimental work with animals has been done on this aspect. For this reason it was thought to be appropriate to undertake in the present work an investigation of this particular question.

The macroscopic, roentgenologic and microscopic findings in the present experiments with arthrotomy and capsulectomy (Group III) are shown in Table 4.

*Macroscopic changes* — In the specimens taken 5, 10 and 15 days after the operation a considerable haematoma was found in the joint and its neighbourhood. Macroscopically, a tissue resembling the synovialis had partially developed already ten days after the operation, at the later examinations the surroundings of the articular capsule consisted of hard cicatrized tissue. In the specimen of the 110th day the joint was luxated. In those taken 120, 130 and 240 days after the interference, there was unevenness of the articular cartilage itself. In the animal sacrificed after 100 days the lig. teres had become reattached. No changes were observed in any of the other cases. From 120 days onwards the joint had a restricted range of motion.

*Roentgenologic changes* — Only two of the cases displayed changes observable in the x-rays, namely luxation after 110 days with a femoral head of considerably smaller size and greater density than the control on the other side and increased density of the femoral head 130 days after the operation.

*Microscopic changes* — 5—15 days. The specimen taken after 15 days contained granulation tissue in the cancellous spaces.

15—45 days. Granulation tissue was found in the cancellous spaces in the specimens of the 20th and 25th days located centrally to the site of



TABLE 4  
Arthroscopy and capsulectomy (Gr 3 L -  
Examination results

| Time of examination<br>days since operation | Macroscopic findings               |           |             |                       | Roentgenologic findings |                                 |                                      |            | Cartilage    |           |
|---|------------------------------------|-----------|-------------|-----------------------|-------------------------|---------------------------------|--------------------------------------|------------|--------------|-----------|
|   | Unevenness of<br>articular surface | Adhesions | Dislocation | Lig. teres reattached | Narrowed joint space    | Changed form of<br>femoral head | Increased density of<br>femoral head | Unevenness | Degeneration | Cartilage |
| 5   |                                    |           |             |                       |                         |                                 |                                      |            |              |           |
| 10  |                                    |           |             |                       |                         |                                 |                                      |            |              |           |
| 15  |                                    |           |             |                       |                         |                                 |                                      |            |              |           |
| 20  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 25  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 30  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 35  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 40  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 45  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 50  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 60  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 70  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 80  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 90  |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 100   |                                    | +         |             | +                     |                         |                                 |                                      |            |              |           |
| 110   |                                    | +         | +           |                       |                         | +                               | +                                    | +          |              |           |
| 120   | +                                  | +         |             |                       |                         |                                 |                                      | +          |              | +         |
| 130   | +                                  | +         |             |                       |                         |                                 | +                                    | +          |              | +         |
| 140   |                                    | +         |             |                       |                         |                                 |                                      |            |              | +         |
| 150   |                                    | +         |             |                       |                         |                                 |                                      |            |              |           |
| 180   |                                    | +         |             |                       |                         |                                 |                                      |            |              | +         |
| 240   | +                                  | +         |             |                       |                         |                                 |                                      | +          |              | +         |
| 360   | No specimen                        |           |             |                       |                         |                                 |                                      |            |              |           |



Epiphyseal cartilage was observed in seven instances among the specimens from the period of 5—50 days and not once after 50 days. On the 20th, 25th, 30th and 35th postoperative days ossification of the epiphyseal line was found to be farther advanced on the operated side than in the corresponding control.

*Autoradiographic changes* — One specimen 240 days after the operation was subjected to autoradiographic study. It revealed uniform deposition of radio-phosphorus in the bone tissue and in the richly vascularized granulation tissue as was distinctly seen in the specimen of the operated left side at the point where granulation tissue was present in the cancellous spaces.

*Micro-radiographic changes* — Micro-radiography was also applied to the specimen examined by autoradiography, i.e. that derived from the animal sacrificed after 240 days. It was found to contain only normal bone.

Necrosis of the bone was recorded in 5 of the 22 operated rabbits.

#### COMMENTS

Comparison of the results elicited in this group by capsulectomy with those in the basic experiments consisting of arthrotomy, division of ligaments and temporary luxation of the femoral head (Group I) revealed that necrosis of the bone developed here at a much later time and occurred only once more frequently. Changes in the articular cartilage were considerably less frequent than in Group I.

Rather little experimental work has been done on the effect of capsulectomy in experimental animals although it is true that KISTLER (1934 and 1936) observed degenerative changes in the bone caused by this interference in young animals. In the present investigation such changes were noted only once in the initial stage but they were distinctly present at a later time between the 120th and 180th days. The low incidence of bone necrosis particularly in the early stage is thought to imply that the cancellous circulation carries greater significance in rabbits.

According to the clinical investigations of BANKS (1941) and of STEWART and MILFORD (1954) damage to the capsular vessels has an important role in the development of necrosis of the bone following luxation. Ample reference has also been made to their significance in fractures of the neck of the femur. In the present work, however, there is much evidence to the effect that in rabbits at least the capsular circulation is not an essential factor, seeing that necrosis of the bone was encountered infrequently only.

If any significance is to be attributed to it, this will be only later when the new capsule hardens and increases in thickness with increasing age and moreover produces distinct impairment of function

Summarizing what has been stated in the foregoing, it can be said that the macroscopic changes were rather insignificant, with the exception of haematoma in the surroundings and of later cicatrization. The roentgenologic changes were likewise negligible, apart from one instance of luxation of the femoral head and of increased density observed in two cases. Neither were any marked microscopic changes noted even in the case presenting luxation. The luxation must therefore have occurred relatively late. Un-evenness of the articular cartilage was microscopically established in four instances. The granulation tissue reaction of the cancellous spaces was low in intensity and indications of necrosis of the bone were not encountered before 120—180 days had passed from the operation, altogether in four instances (except once already on the 35th day). The autoradiographic study showed uniform deposition of radio phosphorus in the normal bone tissue. The structure of normal bone tissue was visible in the micro radiograph.

*Changes occurred in my opinion, in surprisingly small numbers in this experimental group. Observations worth noting are that, with the exception of the fairly rapidly developing granulation tissue reaction the changes in the bone did not begin until after 110 days showing then some correlation with the changes in the articular cartilage. This was obviously attributable to the reduced functional activity due to hardening of the new capsule.*

## ARTHROTOMY AND NEARLY TOTAL OSTEOTOMY OF THE NECK OF THE FEMUR

### EARLIER INVESTIGATIONS

In the experimental group to be considered next the neck of the femur was subjected to osteotomy stopping short of its complete division. But this operation is virtually equivalent to complete fracture of the neck seeing that according to LEMOINE (1957) the point chosen for the osteotomy involves essentially all vasculature supplying blood to the femoral head in rabbits and it seems appropriate to review also the literature referring to such fractures. It was technically preferable to leave a narrow bridge between the fragments of the femoral neck because fixation would have been less secure if the bone had been completely divided.

*Clinical and experimental investigations* — In respect of fractures of the neck of the femur the following incidences among others have been reported for aseptic necrosis as complication of these fractures in spite of operative treatment. EYRE-BROOK and PRIDIE (1941) 14.7 per cent. WATSON-JONES (1942) 30 per cent with operative reduction and 15 per cent with extraarticular nailing. D'AUBIGNE and HERTZOG (1943) 11.5 per cent. LINTON (1944) 39.5 per cent and 9.3 per cent with nailing according to SMITH-PETERSEN and according to NYSTROM respectively. BOYD and GEORGE (1947) 33.6 per cent and (1948) 32.6 per cent. BADO (1948) 24 per cent and 22 per cent with fixation by means of a three-winged nail and by means of a screw respectively. RUSSE (1952) 26 per cent. BOHLER and ENDER (1953) 33.85 per cent. SCAGLIETTI (1953) 42.3 per cent with fixation by screw. CHRISTOPHE *et al* (1953) 17.8 per cent. D'AUBIGNE (1953) 70 per cent in subcapital and 30 per cent in collum fractures. NYSTROM (1954) 25 per cent and WEIS and MULLER (1954) 25 per cent. The figures vary according to the length of follow up period and the type of fracture but they serve to emphasize the remarkable significance of aseptic necrosis as a complication after such fractures.

For conservatively treated (repositioned and immobilized) fractures of the same kind equivalent and even higher incidences have been reported. PHEMISTER (1934) 65 per cent. SANTOS (1930) 60 per cent. SCAGLIETTI

(1953) 41·5 per cent and CARRELL and CARRELL (1941) 25 per cent in children

Although the figures are of the same order of magnitude, it has been suggested that the presence of metal would contribute to the risk of necrosis of the bone BRODETTI (1960) and CLAFFEY (1960) studied this question in 16 and 12 cadavers respectively performing in them SMITH PETERSEN'S nailing and investigating the cancellous circulation by means of transversal specimens and microangiography They concluded that the metal nails contributed little to the occurrence of aseptic necrosis of the bone if the upper posterior quadrant of the femoral head was avoided when the nails were placed A similar observation of the insignificant detrimental effect of the nail was already reported in a single case by WISE (1941)

Several investigators e.g. LANGE (1951) and DUBOIS (1953), stress the importance of circumstances associated with the fracture itself in the aetiology of aseptic necrosis while McELVENNY (1960) still placed particular weight on the essential significance of careful and accurate nailing as did the above mentioned and other authors in recent years Moreover he suggested that the sole true blood supply to the avascular femoral head comes from the cancellous part of the bone while most of the other authors stress the important part played by the capsular and periosteal circulations

*Animal experiments* — BON (1923) produced in six adult dogs fracture of the medial neck of the femur which he fixated by means of a plaster cast The animals were sacrificed 35—100 days later Complete necrosis of the bone was found in two cases partial necrosis in two others coxa vara in one and complete bony repair of the fracture in one case Total necrosis developed when also the lig. teres had been completely divided HENRY (1931) performed on 15 dogs proximal osteosynthesis after complete intracapsular transverse osteotomy In one instance avascular necrosis resulted in the femoral head

COMPÈRE and WALLACE (1942) instituted a comparison between different therapeutic procedures in fractures of the neck of the femur in 38 dogs they examined 21 of the animals and found in 33·3 per cent avascular necrosis if the fracture had been reduced and fixated well otherwise in 66·6 per cent and in untreated cases in 75 per cent TOVEY as cited by GALLIE (1956) produced subcapital fracture in dogs cut off the blood supply and fixated the fracture by means of a screw obtaining a good result in 100 per cent

BONFIGLIO (1954) investigated in 15 adult dogs the repair of subcapital fracture of the neck of the femur and the influence exerted on it by bone

transplantation. The animals were sacrificed after periods from 3 weeks to one year. He found that fixation produced bony consolidation but did not reduce the occurrence of avascular necrosis. Necrotic areas were still encountered in the bone after 3—4 months although the fracture itself had already healed. As occurs also in bone transplants, the bone died in a few days but did not lose its osteocytes until 2—3 weeks after transplantation.

HARRIS and HOBSON (1956) using male rabbits aged 5—6 weeks performed operative epiphyseolysis which they refixed with a Kirschner wire. The first series comprised 45 rabbits which were sacrificed after 1—6 weeks. The authors reported avascular necrosis of the bone and deformation of the femoral head. In another series they detached the epiphysis and observed more rapid revascularization. LANGENSKIÖLD and SARPIÖ (1960) removed the medial epiphysis of young rabbits obtaining *coxa vara*.

BOBECHE and HARRIS (1960) used adult rabbits in their investigation of the comparability of the histologic and roentgenologic findings in aseptic necrosis. They produced necrosis of the bone in 80 rabbits by exploration of the hip through a posterolateral incision and division of the lig. teres in addition to which they divided the neck of the femur with an electric saw. The fracture was then fixed with a Kirschner wire. Necrosis followed already in four days. Repair of the fracture occurred within 10—20 days the femoral head being regenerated so that degeneration of the articular surface or collapse of the femoral head did not occur in a single instance.

MÜLLER (1924) studied traumatic epiphyseolysis in young dogs, cats and rabbits eliciting only transient changes. The animals were sacrificed after 7—47 days and no regeneration had come under way during this period. LAURENT (1959) removed the greater trochanter of some five day-old rabbits producing typical valgus deformity of the femur.

It is seen that by inflicting various kinds of injury to the epiphyseal cartilage aseptic changes of bone have been produced in the femoral head in young animals and that by experimental fracture of the neck of the femur variable changes of the bone of a corresponding kind have been induced in adult animals.

#### OWN INVESTIGATIONS

This part of the present investigation has also been considered indicated, for the reason that the operation involved here damaged the cancellous circulation more directly than any other procedure employed in this work.





The results of the macroscopic roentgenologic and microscopic examinations in the experiments with arthrotomy and nearly total osteotomy of the neck of the femur (Group IV) are shown in Table 5

*Macroscopic changes* — Very slight changes were observed here merely greater than normal firmness of the articular capsule and its environment being noted. In one instance 130 days after the operation the lig. teres was reattached. On cutting samples with the electric saw the neck of the femur was found to be of normal hardness. Distinct infection of the joint was observed once and the femoral head was entirely destroyed in this case. This experimental animal was discarded and another was operated to replace it. Complete fracture was recorded once after 25 days and distinct pseudoarthrosis of the neck of the femur was seen on the 240th day.

*Roentgenologic changes* — Dislocated fracture of the neck of the femur was seen 25 days after the operation (Fig. 20).

The head of the femur appeared equal in density to that on the intact right side. In the animals examined 35, 40, 70 and 80 days after the operation the femoral head was increased in density in addition to which it was flattened on the 35th day. On the 240th day pseudoarthrosis of the neck of the femur was noted on the left side and the femoral head appeared less dense (Fig. 21).

No changes were observed in any other x rays in this group.

*Microscopic changes* — In this connection the report shall be limited to changes that occurred in the femoral head itself disregarding those seen at the point of fracture or associated with the repair of the fracture.

5 — 15 days. In the specimen of the 7th day the articular cartilage was uneven, the nuclei were absent at many points. The femoral head was flattened, the bone marrow poor in cells and in the bone itself there were areas poor in nuclei (Fig. 22). The specimen of the 15th day displayed a thin articular cartilage and areas poor in nuclei in the spongy bone (Fig. 23).

15 — 45 days. In the specimen of the 25th day the articular cartilage was thin, the nuclei were fewer in some areas of the spongy bone and entirely absent in others. In the specimen of the 30th day the articular cartilage had almost entirely disappeared and was replaced by fibrous tissue and fibrous cartilage. The bone tissue was broken in structure, the cancellous spaces were rich in granulation tissue and osteoclasts and osteoblasts were encountered in abundance. After 35 days foamy degeneration of fat cells and some giant cells were present in the bone marrow but



Fig 70 X ray 25 days after arthrotomy and nearly total osteotomy of the neck of the femur. Dislocated fracture of the neck of the femur. The femoral head appears equal in density to that on the right (control) side.



Fig 71 X ray 240 days after arthrotomy and nearly total osteotomy of the neck of the femur. Pseudo-fracture in the neck of the left femur. The head of the femur is denser in comparison with the right side.



Fig 22 Micrograph 5 days after arthrotomy and nearly total osteotomy of the neck of the femur. The femoral head is flattened, the bone marrow poor in cells, and in the bone itself there are some cell poor areas — Weigert van Gieson  $\times 10$



Fig 23 Micrograph 15 days after arthrotomy and nearly total osteotomy of the neck of the femur. Areas poor in cells are present in the bone under the articular surface — Weigert van Gieson  $\times 100$

Fig 24 Micrograph 50 days after arthrotomy and nearly total osteotomy of the neck of the femur. The cancellous spaces are filled mainly with granulation tissue there is slight occasional depletion of nuclei in the spongy bone — P.A.S.  $\times 10$



Fig 25 Dec 11 micr gr pb n Fig 24 Cancellous spaces very rich in granulation tissue but no osteogenesis — P.A.S.  $\times 10$



*Fig. 6 Micrograph 80 days after arthrotomy and nearly total osteomyelitis of the neck of the femur. The articular cartilage is mostly worn off down to the bone. Scarciness of nuclei is observed in the underlying bone — Weigert stain (100)*

there were no changes in the articular cartilage. In the specimen taken on the 40th day the articular cartilage was frayed in spots and its surface was poor in cells. There was some granulation tissue at a few points in the cancellous spaces as was also in the specimen of the 45th day, but the latter showed no changes in the articular cartilage.

**45 — 90 days.** The articular cartilage in the specimen of the 50th day was frayed and then worn down in some places and poor in cells. The cancellous spaces were well filled rich in granulation tissue and there was slight depletion of nuclei at some points in the bone (Figs 24 and 25). In the specimen of the 70th day no changes were observed in the articular cartilage but there was some granulation tissue in the cancellous spaces on one side of the femoral head. The articular cartilage in the specimen of the 80th day was mostly worn off but only a small quantity of granulation tissue was encountered in the cancellous spaces. There was scantiness of nuclei in the bone underlying the articular cartilage (Fig. 26). The specimen of the 90th day revealed slight unevenness of the articular cartilage and some granulation tissue in the cancellous spaces on one side of the femoral head but no osteogenesis and no necrosis of bone.

Fig. 24 Micrograph 50 days after arthrotomy and nearly total osteotomy of the neck of the femur. The cancellous spaces are filled mainly with granulation tissue; there is slight occasional depletion of nuclei in the spongy bone.  
— P.A.S.  $\times 10$

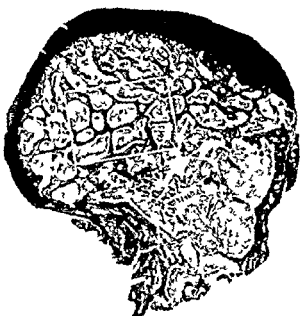


Fig. 25 Micrograph 50 days after arthrotomy and nearly total osteotomy of the neck of the femur. The cancellous spaces are very rich in granulation tissue, but no osteogenesis is seen.  
— P.A.S.  $\times 50$

## COMMENTS

Comparing the changes found in this experimental group with those obtained after the basic operation, i.e. arthrotomy division of lig. teres and temporary luxation of the femoral head (Group I) the changes were found to be lesser in degree in the group now under discussion though necrosis of the bone was encountered once more in this group. In this group, necrosis of the bone occurred at an earlier time and the later specimens contained granulation tissue indicating regeneration. The changes in the articular cartilage too were less marked in this group.

In experiments carried out previously with adult dogs aseptic necrosis of bone followed fractures of the neck of the femur in equal frequency as in man (e.g. BORN 1923, HENRI 1931 and COMPERE and WALLACE 1942). According to some investigators (BONIGLIO 1954, TOVEL 1956 and BOBLICH and HARRIS 1960) aseptic necrosis was a transient phenomenon as also HUSTLER (1936) has shown with a few adult rabbits. In the present investigation repair of the osteotomy failed to occur in two cases only although it has to be kept in mind that the osteotomy performed here while certainly comprising the most essential portion of the femoral neck as regards the blood supply of the femoral head, was not complete. Of course, this investigation did not aim at a study of the repair of osteotomized bone but rather at an understanding of the changes of bone and articular cartilage occurring in the femoral head.

Necrosis of bone and also degeneration of the articular cartilage were observed at a comparatively early stage. In the later specimens on the other hand the histologic picture was dominated by the occurrence of granulation tissue indicative of regenerative ability. Thus in these instances the necrosis of bone encountered 5 times was a transient phenomenon associated with the repair of the fracture as was already shown by BARTH (1893).

As a rule authors have stressed damage caused to the capsular vessels as an agent producing necrosis of bone in fractures of the neck of the femur but McCLIVLNNY (1960) constitutes an exception in stating, in his clinical studies that the cancellous circulation is more important. The present investigation with rabbits supports his opinion. The blood vessels supplying nutrition were severed in the operation but as the osteotomized fragments were satisfactorily retained in their proper positions, a cancellous circulation was rapidly established and good consolidation of the osteotomy ensued with exception of the case in which the femoral head became detached and complete necrosis of the bone followed within 25 days. In

the case with pseudoarthrosis, after 240 days no bone changes were observed

The regenerative power of the bone tissue in the experiments of this group was nicely demonstrated also by the fact that hardly any scantiness of cells was noted in the bone marrow even when there were changes in the bone. Although the blood supply of the femoral head had been destroyed the slight changes in the bone healed rapidly because, apart from the direct instrument induced trauma no pressure effect altered static conditions or remarkable reduction of function were present in this experiment

The decrease in the articular changes with time finds its explanation in the mere operative trauma seeing that no actual degenerative changes occurred simultaneously with necrosis of the bone except in the very first specimens. Owing to the more radical operative intervention the direct trauma was heavier here than in the preceding groups. After five days degenerative changes were noted in the cartilage as well as in the bone but also BOBECHO and HARRIS (1960) have been able to observe such rapid changes. Further corroboration of this is the fact that necrosis of bone had already fully developed in the specimen of the 15th day.

The roentgenologic changes observed in this group show no fully convincing correlation with the histologic picture inasmuch as the roentgenologically noted increased density of the femoral head which occurred in six instances was concomitant with microscopically observed necrosis of the bone in one case only.

Summarizing what has been stated in the foregoing it seems justified to say that the macroscopic changes were insignificant, with the exception of one completely dislocated fracture of the neck of the femur and one case of pseudoarthrosis. The changes were also visible roentgenologically. Moreover increased density was revealed by the x rays in four specimens while the changes otherwise were insignificant in amount. Only the first specimen showed the osteotomy on macroscopic examination. The said four specimens had granulation tissue in the cancellous spaces though in scanty amount.

Microscopically degenerative changes in the articular cartilage itself were noted between the 30th and the 120th day simultaneously a distinct though mostly slight granulation tissue reaction could be noted in the cancellous spaces. Necrosis of the bone was encountered only 5 times in the early specimens of the group and showed an obvious transient character. The autoradiographic changes were equal on both sides revealing



deposition of radio-phosphorus in the bone but not in the articular cartilage. No differences between the left (operated) and right (control) hips were observed in the microradiographs.

On the strength of the present experiments it is my opinion that the changes of an obviously transient character encountered in this experimental group were mainly associated with the general phenomenon of fracture repair and give evidence of the regenerative power of bone tissue.

## ARTHROTOMY AND TIGHT LIGATION OF THE NECK OF THE FEMUR WITH STEEL WIRE

### EARLIER INVESTIGATIONS

*Animal experiments* — Without actually injuring the bone NUSSBAUM (1923 1923) excised the synovialis of the neck of the femur in young dogs and divided the lig. teres producing a histologic picture resembling that of chondrosis coxae juvenilis. BERGMAN (1927) repeated the experiments of NUSSBAUM (and of MULLER 1924) with a few rabbits confirming the previous findings.

BENTZON (1926) injected alcohol close to the upper epiphysis of the femur in young rabbits and obtained a histologic condition resembling coxa plana. INTROZZI (as cited by LERICHE 1934) produced in 44 rabbits aged  $3\frac{1}{2}$  to 4 weeks flattening and local necrosis of the femoral head by various means such as injection of alcohol and ammonia and injury to the lig. teres.

MILTNER and HU (1933) repeated BENTZON's and INTROZZI's experiments but were unable to confirm their results. On the other hand in eleven rabbits aged  $2\frac{1}{2}$  months which were sacrificed after observation periods of  $2\frac{1}{2}$ — $4\frac{1}{2}$  months they observed necrosis of cartilage and necrotic changes in the underlying bone after ligation of lig. teres, peeling of the periosteum and application of a silk ligature to the neck of the femur.

STEWART (1933) peeled the neck of the femur in young and adult rabbits and in adult dogs, sacrificing them after 7—120 days and found levelling of the femoral head and necrosis of the bone. However he did not consider these changes resemblant of coxa plana. KISTLER (1936) applied a silk ligature to the neck of the femur in rabbits aged 4 and 30 days obtaining changes indicative of necrosis of the bone in the first mentioned but not in the latter.

JAKOBSEN (1937) observed in connection with cup arthroplasties which he performed also in rabbits that total atrophy of the head and neck of the femur resulted in four cases out of ten. KETTUNEN (1958) mentioned in a report on his preliminary tests concerning skin arthroplasty with cats

that he had to abandon the practice of drilling holes through the neck of the femur intended to achieve attachment of the skin flap because aseptic necrosis followed from it. HOOVER and COVENTRY (1961) observed in their skin arthroplasties in dogs that aseptic necrosis developed only twice in 57 operations although the neck of the femur was loosely ligated for attachment of the skin flap.

### OWN INVESTIGATIONS

From the above it is noted particularly that as a rule only very young experimental animals have been studied in the respect in question. Older animals have been used only for reference in single instances. In view of this the present investigation was thought to be appropriate.

The macroscopic roentgenologic and microscopic findings in the experiments with tight ligation of the neck of the femur with steel wire (Group V) are shown in Table 6.

*Macroscopic changes* — Slight unevenness of the femoral head was observed in the specimens taken 10, 20, 25, 30, 35, 40, 45 and 120 days after the operation. The steel wire passed through the trochanter was covered by a thick mass of scar tissue in all except the specimens of the 5th, 10th and 20th days. The steel wire had also produced a strong callus reaction in the neck of the femur (Fig. 30). When the specimen was being detached with the electric saw the neck of the femur was invariably found to be quite soft. Luxation of the femoral head was noted in two instances, namely in the animals sacrificed on the 100th and 120th days.

*Roentgenologic changes* — 10 days after the operation the left femoral head had a denser appearance than that on the right (control) side. On the 40th day it was flattened and of greater density than the control, as also was the case on the 45th day. On the 60th day a distinct radiolucent cavity was observed in the centre of the femoral head. At 70, 80 and 90 days slight increases in density and deformities were noted in the femoral heads. In the specimens of the 100th and 120th days the femoral head was luxated and the steel wire had almost sheared off the femoral head, which was of greater density than that on the control side. There was osteoporosis in the neck of the femur 110 days after the operation and the femoral head appeared less dense (Fig. 28). No changes were observed in any other x rays.

*Microscopic changes* — 5 — 15 days. In the specimen of the 10th day the articular cartilage was more poorly stained in spots and it had lost its nuclei. The margins of the spongy bone under the articular car-

TABLE 6

*Arthrotomy and tight ligation of the neck of the femur with a steel wire  
(Group VI) Examination results*

| Time of examination<br>days since operation | Macroscopic findings               |           |             |                       | Roentgenologic findings |                                 |                                      | Microscopic findings |              |                    |          |          |           |
|---|------------------------------------|-----------|-------------|-----------------------|-------------------------|---------------------------------|--------------------------------------|----------------------|--------------|--------------------|----------|----------|-----------|
|   | Unevenness of<br>articular surface | Adhesions | Dislocation | Lig. teres reattached | Narrowed joint space    | Changed form of<br>femoral head | Increased density of<br>femoral head | Cartilage            |              | Bone               |          |          |           |
|   |                                    |           |             |                       |                         |                                 |                                      | Unevenness           | Degeneration | Granulation tissue | New bone | Necrosis | Epiphysis |
| 5   |                                    |           |             |                       |                         |                                 |                                      |                      |              |                    |          |          | +         |
| 10  | +                                  |           |             |                       |                         |                                 | +                                    |                      | +            | +                  | +        |          | +         |
| 15  |                                    |           |             |                       |                         |                                 |                                      |                      |              |                    |          |          | +         |
| 20  | +                                  |           |             |                       |                         |                                 |                                      | +                    | +            | +                  | +        |          | +         |
| 25  | +                                  | +         |             |                       |                         |                                 |                                      | +                    | +            |                    |          | +        | +         |
| 30  | +                                  | +         |             |                       |                         |                                 |                                      | +                    | +            | +                  | +        | +        | +         |
| 35  | +                                  | +         |             |                       |                         |                                 |                                      | +                    | +            | +                  |          | +        |           |
| 40  | +                                  | +         |             |                       |                         | +                               | +                                    | +                    | +            | +                  | +        | +        | +         |
| 45  | +                                  | +         |             |                       |                         | +                               | +                                    | +                    | +            | +                  |          | +        |           |
| 50  |                                    | +         |             |                       |                         |                                 |                                      |                      |              | +                  | +        | +        | +         |
| 60  |                                    | +         |             |                       |                         | +                               | +                                    | +                    |              | +                  | +        |          |           |
| 70  |                                    | +         |             |                       | +                       | +                               |                                      | +                    |              |                    |          | +        |           |
| 80  |                                    | +         |             |                       |                         | +                               | +                                    |                      |              |                    |          | +        |           |
| 90  |                                    | +         |             |                       |                         | +                               | +                                    |                      |              |                    |          | +        |           |
| 100   |                                    | +         | +           |                       | +                       | +                               | +                                    | +                    | +            | +                  | +        | +        |           |
| 110   |                                    | +         |             |                       |                         | +                               |                                      | +                    | +            |                    |          | +        |           |
| 120   | +                                  | +         | +           |                       | +                       | +                               | +                                    | +                    | +            | +                  | +        | +        |           |
| 130   |                                    | +         |             |                       |                         |                                 |                                      | +                    |              | +                  |          | +        |           |
| 140   |                                    | +         |             |                       |                         |                                 |                                      |                      |              |                    |          |          |           |
| 150   |                                    | +         |             |                       |                         |                                 |                                      |                      |              |                    |          |          |           |
| 180   |                                    | +         |             |                       |                         |                                 |                                      |                      |              |                    |          |          |           |
| 240   | No specimen                        |           |             |                       |                         |                                 |                                      |                      |              |                    |          |          |           |
| 360   | +                                  |           |             |                       |                         |                                 |                                      | +                    | +            |                    |          | +        |           |



*Fig. 2. X ray 110 days after ligation of the neck of the femur with steel wire. On the operated side there is a defect in the femoral neck and the head of the femur also appears to be less dense.*

tilage contained granulation tissue, osteoblasts and new bone (15-29)

15-45 days. The specimen taken on the 20th day showed unevenness of the articular cartilage which was thin and poor in cells along one margin (Figs 30, 31 and 32). In the cancellous spaces in the middle of the femoral head there was granulation tissue as well as osteoblasts and osteoclasts (Fig. 33). In the specimen of the 25th day the cells in the basal part of the articular cartilage were anuclear for the greater part. The same changes were observed in the specimen of the 30th day where furthermore the articular cartilage was frayed and worn off in spots as also was the case after 35 days.

In the specimen of the 40th day the hyaline cartilage was partly converted into fibrous cartilage and its surface was uneven. At the boundary between bone and cartilage the cartilage cells were fairly large in size. At 45 days the articular cartilage was uneven and here and there worn off down to the bone in addition to which it had been replaced in some places by fibrous tissue and fibrous cartilage. Elsewhere it was thickened with confused arrangement and greater number of cells. Extensive necrosis of the bone was present in all other specimens of this group.



*Fig 29 Micrograph 10 days after ligation of the neck of the femur with steel wire The articular cartilage has lost its nuclei at certain points and is uneven Beneath it on one side are seen osteoblasts and already new bone — Weigert van Gieson 100*



*Fig 30 Micrograph 20 days after ligation of the neck of the femur with steel wire The steel wire has penetrated deep into the neck of the femur and produced a strong callus reaction in its neighbourhood — Weigert van Gieson  $\times 10$*



Fig. 31 Micrograph 20 days after ligation of the neck of the femur with steel wire (same specimen as in Fig. 30). Abundant cellular tissue around the steel wire and granulation tissue in the adjacent articular spaces. — Weigert van Creson.  $\times 40$

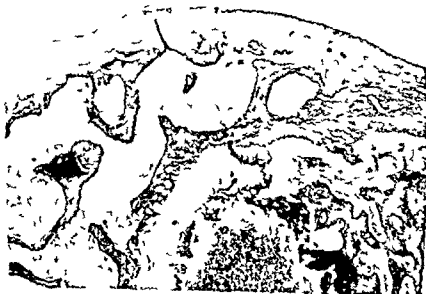


Fig. 32 Micrograph 0 days after ligation of the neck of the femur with steel wire (same specimen as in Fig. 30). Thin articular cartilage poor in cells. — Weigert van Creson.  $\times 40$



Fig 33 Micrograph 20 days after ligation of the neck of the femur with steel wire (same specimen as in Fig 30) In the cancellous space granulation tissue and incipient formation of new bone On the left new bone is apposed on the old bone and osteoblasts are seen in abundance — Weigert & an Gieson  $\times 500$



Fig 34 Micrograph 25 days after ligation of the neck of the femur with steel wire Unten articular surface and empty cancellous spaces the bone tissue and epiphyseal cartilage are anuclear — P.A.S.  $\times 40$



Fig 35 Micrograph showing specimen from the right (control) side of the same animal as in Fig 34. No signs of degenerative changes in the cartilage or bone.  
— P.A.S.  $\times 40$



Fig 36 Micrograph 30 days after ligation of the neck of the femur with steel wire. The spongy bone is poor in spots and granulation tissue is present in the cancellous spaces.  
— P.A.S.  $\times 100$





Fig. 41 Micrograph of the control side opposite to the operated side in Fig. 40. The spongy bone is nucleated and the number of cells in the bone marrow is normal — P.A.S.  $\times 100$

In the specimen of the 25th day the cancellous spaces were empty and the bone tissue itself was poor in cells (Figs. 34 and 35) but in the specimens of the 30th day (Figs. 36—39) and of the 35th, 40th and 45th days granulation tissue was noted in the cancellous spaces in considerable quantity. In those of the 30th and 40th days there were moreover osteoblast and osteoclast agglomerations at several points and scantiness of nuclei in the bone tissue.

45 — 90 days In the specimen of the 50th day extensive anuclear necrotic areas of bone were seen, but no changes in the articular cartilage. The granulation tissue had increased in the cancellous spaces and incipient osteogenesis was seen here and there. The specimen taken on the 70th day showed occasional unevenness and wear of the articular cartilage but in the specimens of the 80th and 90th days the articular cartilage was fairly smooth. In these specimens areas poor in nuclei were encountered in spots in the spongy bone but no granulation tissue and no osteogenesis was seen. The Alcian blue stained sample of the 90th day contained numerous anuclear areas that had acquired only a light red stain.

90 — 360 days In the specimens derived from the animals sacrificed on the 100th and 120th days in which also the effect of luxation was present, the articular cartilage was found to have largely been worn off

or replaced by fibrous cartilage or fibrous tissue. In the corresponding samples of the cancellous spaces granulation tissue was found in abundance and there were osteoblasts, osteoclasts and areas without nuclei.

The specimen of the 110th day presented a particularly broken articular surface with a top layer poor in cells and areas poor in nuclei in the underlying spongy bone. In the specimen of the 130th day the articular cartilage was uneven and the nuclei were absent in the spongy bone over an extensive area. The cancellous spaces were rich in granulation tissue (Figs. 40 and 41). In the specimens taken on the 140th, 150th and 180th days again no changes were observed.

The specimen of the 360th day had a bone tissue that was broken up and poor in nuclei; the articular cartilage was occasionally even but its greater part was destroyed and replaced by fibrous tissue or fibrous cartilage.

Epiphyseal cartilage was observed in eight instances among the specimens from the period between the 5th and 50th days but not at any later time. In the specimens of the 5th and 25th days ossification of the epiphyseal line was farther advanced on the operated side.

*Autoradiographic changes* — An autoradiographic study was made of the two specimens taken on the 80th and 90th days. In the first mentioned instance the distribution of radio-phosphorus was found to be less uniform on the left (operated) side but it indicated considerably greater deposition. In the specimen of the 90th day the radio-phosphorus was distributed over the entire area of bone tissue also on the left side however so that less deposition had occurred in the areas of necrotic bone (Fig. 42).



Fig. 42. Contrast autoradiographs obtained with radio-phosphorus of the specimen taken 90 days after arthrotomy and ligation of the neck of the femur with steel wire. Comparison shows apparently less deposition at the point of bone necrosis (indicated by an arrow) on the operated left side.

*Microradiographic changes* — The same specimens which were used for autoradiographic studies were also subjected to microradiographic examination that is the specimens of the 80th and 90th day. A slight difference was noted here between the two sides with respect to lower x ray absorption by the surrounding areas than by necrotic bone.

Necrosis of the bone was observed in 14 instances among the 22 operated rabbits although in two of them also the effect of luxation was present.

#### COMMENTS

When the effects of the basic operation that is of arthrotomy division of lig. teres and temporary luxation (Group I), are eliminated from the results reported above it will be seen that the degenerative changes in the cartilage and the necrotic changes in the bone encountered in this experimental group were of a very high degree exceeding those in any other group concerned in this work.

As has been learned from earlier investigations necrosis of the bone can be produced in the femoral head by causing damage to the neck of the femur in various ways. MILNER and HU (1933) employed the method described here but their experimental animals were young rabbits of 2½ months age. KISTLER (1936), too, obtained the most marked changes of bone necrosis in his experiments with rabbits by this procedure, although also his animals were young. In the present work already at a comparatively early stage after ten days a change was found in the articular cartilage from which also the nuclei had partially disappeared.

According to previous reports the change develops at a fairly slow rate in the articular cartilage (HALDEMAN 1938, CARLSON 1957) and it may therefore be one of preoperative origin. This is not likely, however, seeing that not a single one of our cases presented a similar change on the other side which served as control.

Degeneration of the cartilage occurred twice although no necrosis of bone was observed and the opposite was noted to have occurred five times. It is conceivable that in the first mentioned instances the change is visible sooner in the cartilage while the bone also has obviously been in the process of necrosis though it is not yet anuclear. The necrosis of bone was then already evident in the subsequent specimens. When necrosis of bone was observed 25 days after the operation and the cancellous spaces were at the same time found to be empty, this was a sign of fairly complete necrosis of the bone. The regenerative ability however was

strong enough so that granulation tissue could be seen already in the following specimens between anuclear areas of bone. The time at which necrosis of the bone became demonstrable was also in this experimental group fully consistent with observations in earlier investigations (AUCHAUSEN 1922 1926 1928, WOLLENBERG 1928 AUCHAUSEN and BERGMAN 1937).

In the specimen taken on the 80th day no change of the cartilage was encountered. Obviously no change had been produced in them, the synovial fluid and functional activity of the joint maintaining a normal condition of the cartilage or regeneration had already occurred before the specimen was taken.

In the specimens of the 100th and 120th days in which also the effect of luxation was present massive necrosis of bone and destruction of the cartilage had taken place. Consequently owing to the combined effect these specimens cannot be used as representatives of this group in comparisons with the other groups. In the specimens taken on the 140th 150th and 180th days in which no changes were observed the cancellous blood supply of the bone obviously played already an essential part and the changes that would have been noted at an earlier time had already been repaired.

In also this group the autoradiographic studies with radio phosphorus furnished indications to the effect that the uptake of phosphorus is greater by new bone than by normal bone which inferentially implies that more active metabolism and also more profuse vascularization exists there as has been quite generally observed. Microradiography confirmed the results obtained in the roentgenographic examinations in respect of distribution of the mass making it possible to observe the reaction in the areas adjacent to the necrotic bone.

In every case in this group a strong reaction of the surrounding tissues and formation of cartilaginous callus at the point involved had been caused by the pressure atrophy produced by the wire. Considering the investigations of LEMOINE (1957) this in itself may have impeded the circulation at the surface of the bone, and the cancellous circulation was not able to compensate for this loss until at a later time.

Summarizing what has been stated in the foregoing it may be said that in addition to the scar tissue that had been formed around the wire, the macroscopic findings consisted of the observation that the neck of the femur was softer than normal. Roentgenologically, increased density of the femoral head was noted in eight instances and osteoporosis of the neck of the femur or greater radiolucency in two cases.

The microscopic changes were great already in the specimen taken after 20 days. In the articular cartilages the examination revealed, in addition to degenerative changes, also extensive wear in many specimens. In the spongy bone itself and in the cancellous spaces abundant degenerative changes and fairly complete necrosis of bone developed as early as by the 25th day and in altogether 14 cases. This included, it is true, two cases in which the effect of luxation also was present.

Granulation tissue reaction and osteoblasts and osteoclasts in profuse numbers were observations associated with the preceding, in the Alcian blue staining the new bone was found to acquire a more intensive red stain while necrotic bone showed a lighter red hue than normal spongy bone. In the P A S stain necrotic bone responded less strongly to the staining agent than living bone tissue which stained violet.

Autoradiography revealed a distinct difference in the concentration of radio-phosphorus in the bone on the operated left side and on the right side serving as the control. The effect correlated well with the histologic and microradiographic pictures in that more phosphorus was deposited in the area of osteogenesis and less in necrotic areas than in normal bone.

*According to my observations the changes were greater in this group than in any other experimental group the wire causing considerable pressure atrophy, followed by degeneration and necrosis in the neck of the femur which the cancellous circulation naturally was unable to prevent. The correlation with the changes in the articular cartilage was distinct with mainly simultaneous effects.*

## LIGATION OF THE DEEP FEMORAL ARTERY AND THE OBTURATOR ARTERY

### EARLIER INVESTIGATIONS

*Animal experiments* — BENASSI (1931) ligated the femoral artery and the iliac artery both with and without simultaneous venous ligature in rabbits of varying age but he did not note any alterations in the bone. PEARSE (1928) excised the entire femoral artery of six dogs with no noteworthy detriment a strong collateral circulation having developed within two weeks. OLOVSON (1942) ligated the femoral artery and iliac artery of 23 rabbits and examined the circulation at various times between 24 hours and nine months. He found that collateral paths had been established.

BERGMAN (1927) in his experiments with 13 dogs succeeded by injection of metal powder into the femoral artery in producing necrosis of the bone in the diaphysis of the long bones in six cases but not in the epiphysis. KAHLSTROM *et al* (1939) made unsuccessful attempts to produce necrosis of bone in seven dogs by injection of air into the artery and by keeping the animals in the Trendelenburg position. COLONNA and JONES (1948) showed experimentally with 12 rabbits that necrosis of the bone was produced when a vacuum acted on the bone marrow itself setting free the nitrogen that was present there.

JEMOINE (1957) carried out investigations with 60 rabbits five of them adult clarifying the normal circulation in the rabbit hip by microangiography and by means of impediment caused in different ways to the blood supply of the femoral head. He found that surgical ligation of the most important capsular vessel the anterior circumflex artery resulted in young rabbits in changes resembling osteochondritis juvenilis in man. On the other hand no corresponding change was elicited in three adult rabbits which were sacrificed after 35, 42 and 90 days. He also noted that regeneration takes 90 days in young rabbits and that the actual hypovascularization phase in the femoral head lasts 15 days.

JUDER *et al* (1955) using autopsy material have carried out experimental studies including ligation of the above mentioned arteries. These experi





ments have been reviewed in the chapter dealing with the effects of capsulectomy on p 51

#### OWN INVESTIGATIONS

This part of the investigation was undertaken entirely for an experimental purpose to serve as a reference series

The macroscopic roentgenologic and microscopic findings in the experiments with ligation of the deep femoral artery and of the obturator artery (Group VI) are shown in Table 7

*Macroscopic changes* — No changes were observed in the articular cartilage In the musculature of the trochanteric region there were extensive cicatricial strictures The specimens of the 5th and 15th days showed fairly massive haematomata

*Roentgenologic changes* — No changes were observed

*Microscopic changes* — On the 45th day the articular cartilage was broken up at one point In the other specimens it was even and no pathologic changes were present in it nor were there noteworthy changes in the spongy bone or in the bone marrow

Epiphyseal cartilage was recorded in two instances among the specimens taken between the 5th and 50th days but not thereafter

*Autoradiographic changes* — One specimen taken 90 days after the operation was subjected to autoradiographic study The distribution of radio-phosphorus was similar on both sides and it was observed that the deposition in the bone was uniform (Fig 43)

*Micro-radiographic changes* — One specimen, which also was subjected to autoradiography (specimen of the 90th day) was examined by micro-



Fig 43 Contact autoradiograph obtained with radio-phosphorus of the specimen taken 90 days after ligation of the deep femoral artery and the obturator artery  
The deposition is uniform and equal on both sides

radiography There was no difference between the two sides in respect of distribution of the mass

#### COMMENTS

In conformity with earlier investigations no changes were seen in the femoral head after ligation of the large arteries Obviously, in order that necrosis of bone might be produced by this interference the ligation should be quite local, as was that applied by LEMOINE (1957), and the subjects should furthermore be young individuals

The subject's age and possibly the metabolic processes in the bone are important factors in vascular lesions A disturbance no doubt results more easily in necrosis in young subjects, but on the other hand, as is well known regeneration is more rapid in them

As has been shown by earlier research (COLONNA and JONES 1948), necrosis of bone can be produced by obstructing the blood flow for example by means of air but also then the interference must have immediate reference to the internal circulation

Summarizing what has been stated above it may be said that the macroscopic roentgenologic histologic autoradiographic and microradiographic changes following ligation of the deep femoral and the obturator arteries in the femoral head were practically nonexistent

## DISCUSSION

Each surgical interference concerned in this work is an entity in itself and the results elicited in the respective experiments have therefore been considered separately and in relation to each other in the foregoing. In the following shall be presented the thoughts and inferences which have emerged from my investigation. For the reader's convenience the results of the macroscopic roentgenologic and microscopic examinations are shown once more in Tables 8, 9 and 10 grouped by the methods of investigation.

Cartilage and bone are derivatives of connective tissue. One would therefore be inclined to expect that although they possess additional properties owing to their specialization they would respond in the manner of connective tissue and of other living tissues. For instance they respond slowly to various factors that produce degeneration to the extent that regenerative phenomena such as the formation of granulation tissue may become evident sooner than the actual necrosis itself.

It is well known that a great variety of traumatic circulatory, external and internal factors may exert a degenerative and even a necrotic effect on connective tissue which responds by regeneration. Cartilage and bone are not static tissues; it is a known fact that continuous destruction and rebirth of cells takes place in them. These phenomena are greatly affected by among others hormonal factors but also topical influences such as altered static conditions, direct trauma, reduced functional activity and circulatory conditions have a considerable bearing in this respect.

In the present investigation evidence has come forth to the effect that internal disturbances in the blood supply of bone are able to produce changes in the bone and the cartilage as for example in connection with osteotomy but at the same time this material reveals that such changes are also brought about by the trauma and altered static conditions resulting from dislocation, and by the reduced functional activity consequent on capsulectomy. Pressure applied by means of a steel wire also causes marked changes. Degeneration and necrosis are thus a result of the action of various factors. The literature cites a great number of other factors which

TABLE 8 *Macroscopic findings in the different experimental groups*

| Time of examination,<br>days after operation | Ventricular flaps |  |  | Ventricular wall |  |  | Ligaments of heart |  |  | Aorta |  |  | Coronary arteries |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  | Ligaments of heart |  |  |  |  |  |
|--|-------------------|--|--|------------------|--|--|--------------------|--|--|-------|--|--|-------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--|--|--|
|--|-------------------|--|--|------------------|--|--|--------------------|--|--|-------|--|--|-------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--------------------|--|--|--|--|--|

TABLE 9 Roentgenologic findings in the different experimental groups

[illegible]

TABLE 10 Macropic findings in the different experimental groups

[illegible]

are able to produce changes of this kind and which have not been considered in the present investigation such as heat cold various chemicals and irradiation with x rays

In my opinion the present results also point to the possibility that aseptic necrosis of the bone may not in itself be detrimental under all conditions but that it may be a natural phenomenon conducive to the occurrence of regenerative processes for example in the repair of osteotomized bone

Even interferences as radical as the ligation of blood vessels capsulectomy division of the lig teres and temporary luxation of the femoral head in connection with arthrotomy in rabbits seemed to have comparatively insignificant and transient consequences This would seem to justify the opinion that the cancellous blood supply of the femoral head and neck plays an important role This is understandable enough seeing that the growth of granulation tissue and the regenerative processes commence just in the cancellous spaces It would thus seem that in surgery of the femoral head and the neck of the femur attention should be paid to promotion of the cancellous blood supply This principle has indeed been applied in the therapy of pseudoarthrosis after fracture of the neck of the femur It is also obvious on the other hand that great weight should be placed on the static conditions which should be rendered as favourable as possible since the bone and cartilage which are being formed are greatly dependent on prevailing static conditions

These facts are probably largely responsible for the benefit derived from osteotomy in the treatment of pseudoarthrosis Furthermore the present investigation may be taken to indicate that various traumata may produce degenerative changes and even necrosis of bone and cartilage

It was observed in this work that the degenerative changes in the cartilage and bone and those resulting in necrosis are so correlated that when the cartilage is more directly subjected to trauma as after dislocation it will be damaged first and similar changes in the bone will follow later When the disturbance of the blood supply to the bone is obviously the primary occurrence such as it was when the neck of the femur was tightly ligated with a steel wire both kinds of changes develop nearly simultaneously

The present experiments serve to confirm for their part the prevalent opinion that necrotic bone absorbs x rays more strongly than normal bone in x ray examinations and that new bone has less mass than normal tissue Recognition of necrosis of the bone by autoradiography has proved a difficult problem In the present work too the best indications of degeneration and necrosis of the bone were found to be the degenerative

changes in the nucleus of the osteocyte and its karyolysis, which already were described by AXHAUSEN in 1922. To be sure occurrence of granulation tissue or emptiness of the cancellous spaces already before this may constitute signs of incipient necrosis of the bone.

The observation was made in the histochemical examinations in the present experiments that living bone tissue yields a stronger positive reaction to P A S stain than dead, anuclear bone tissue. According to RISSANEN (1960) the histochemically demonstrable quantity of mucopolysaccharides in connective tissue is reduced in the process of degeneration and in the present work this was found to hold true also for cartilage and bone tissue. On the other hand bone tissue in the process of being formed gives a more intensive P A S reaction as also reported by McMANUS (1948) and HORCHKISS (1948). Alcian blue staining reveals similar changes and by this method new bone acquires a more intensive red stain owing to its higher collagen content. Moreover irregularity of structure was noted in necrosis of the bone of longer duration.

It may be worth while to contemplate continued investigation of the degenerative changes of bone and cartilage by means of enzyme chemical methods which give a rapid reaction as observed by *e.g.*, RAEKALLIO (1961). Even in a contusion wound for instance they reveal clearly observable changes in the distribution of acid phosphatase and leucine amino peptidase as early as a few hours after infliction of the wound.

The practice of referring to the phenomena considered here as avascular necrosis of the bone receives little encouragement from what has been found in the present work. It seems in my opinion that the name employed in the title of this book *aseptic necrosis of bone* should be preferred as it better calls attention to the numerous factors of various kinds responsible for such necrosis.



## SUMMARY AND CONCLUSIONS

The purpose of the present experimental study of the hip joint of the rabbit was to clarify the contribution of various surgical factors to the development of necrosis of the bone

A total of 155 rabbits were used divided into six groups subjected to the following surgical interferences

- Arthrotomy by posterolateral approach division of lig teres and temporary luxation of the femoral head
- As the preceding group but without reduction of the luxation
- Arthrotomy and capsulectomy
- Nearly total osteotomy of the neck of the femur by arthrotomy approach
- Arthrotomy and tight ligation of the neck of the femur with a steel wire
- Ligation of the deep femoral artery and the obturator artery without arthrotomy

In each case the left hip of the rabbit was subjected to the surgical interference while the right hip served as control The periods of observation ranged from 5 to 360 days one animal in each group being sacrificed at each specified date In addition to macroscopic and roentgenologic examination of the operated and control hips specimens were taken from both hips for histologic examination and stained by Weigert van Gieson's haematoxylin method and in some cases with Pentachrome II Alcian blue and periodic acid Schiff staining after EDTA decalcification were also used In eight instances autoradiographs were made by the contact method using radio-phosphorus and the same specimens were also examined by microradiography

Following *arthrotomy, division of lig teres and temporary luxation of the femoral head* quite insignificant macroscopic roentgenologic and microscopic changes were observed However partial necrosis of the bone developed in 4 out of 22 rabbits and numerous specimens revealed unevenness of the articular cartilage and its destruction and replacement by fibrous cartilage and fibrous tissue

In the rabbits operated similarly but with *permanent luxation* there were on the other hand, in addition to definite macroscopic and roentgenologic changes, distinct destruction of the articular cartilage from the very beginning and granulation tissue in the cancellous spaces of the underlying bone. Sixty days and more after the operation there was necrosis of the bone in altogether 11 out of 22 cases. Even then the histologic picture was dominated by granulation tissue reaction and osteoclast and osteoblast activity.

After arthrotomy and *capsulectomy* only insignificant macroscopic and roentgenologic changes were encountered. The articular cartilages showed slight unevenness. The necrotic changes in the bone itself were fairly insignificant, occurring in 5 out of 22 cases.

When *nearly total osteotomy of the neck of the femur* by the arthrotomy approach was performed, repair took place within 10—20 days and there were slight macroscopic and roentgenologic changes. Histologically, degenerative changes in the articular cartilage itself were seen between the 30th and 120th days after the interference. Simultaneously a distinct but scanty granulation tissue reaction was present in the cancellous spaces. Necrosis of the bone was only encountered transiently in 5 out of 22 cases.

Tight *ligation of the neck of the femur* with a steel wire in connection with arthrotomy produced in addition to massive macroscopic changes, increased roentgenologic density of the femoral head between the 40th and 120th days after the operation. The microscopically observable changes were great. By the 20th day the degenerative changes in the articular cartilage were supplemented by complete necrosis of bone in the femoral head with its accompanying regenerative phenomena. Necrosis of the bone resulted in altogether 14 out of 22 cases, although this includes two cases in which also the effect of luxation was present.

Following *ligation of the deep femoral artery and of the obturator artery* without arthrotomy there hardly were any changes.

The macroscopic and roentgenologic changes were well correlated with the microscopic findings. When the articular cartilage degenerated or was destroyed a granulation tissue reaction was produced in the cancellous spaces of the underlying bone. Necrosis of the bone was a frequent consequence; it was slow in developing upon luxation but rapid and complete after ligation of the neck of the femur in connection with arthrotomy. The greater changes were also roentgenologically demonstrable.

The autoradiographic studies with radio phosphorus revealed ample position of phosphorus in the bone, particularly in the new bone, while

necrotic areas of the bone took up phosphorus poorly as also did the cartilage. Microradiography revealed the bone to be less dense than normal bone in actual necrosis of the bone the thickness of the mass is difficult to demonstrate and rather tends to be irregular in its surroundings.

A great variety of causes were deemed to be responsible for the disturbances resulting in degeneration and necrosis of the bone. Parallel to circulatory disturbances an influence in this respect is claimed at least by direct trauma, pressure effect on the articular surface and on the neck of the femur altered static conditions and reduced functional activity. The cancellous circulation was found to surpass the cortical circulation in importance.

Owing to the retained structure of the collagen, living bone tissue acquired a distinctly more intense stain than aseptic, anuclear bone tissue in the P.A.S. and the Alcian blue stainings but the latter remain reliable sign of aseptic necrosis of the bone was the lack of nuclei in the osteocytes. This was usually preceded by scantiness of cells or occurrence of granulation tissue in the cancellous spaces. However confirmation of the early diagnostics of bone necrosis will require further investigation.

The following conclusions seem to emerge from the present investigation.

1 The greatest degenerative and necrotic changes in the cartilage and the bone occurred when the neck of the femur was tightly ligated with a steel wire as a result of pressure by and reaction to the wire and when in addition to arthrotomy and division of ligaments the femoral head was left in its luxated position, as a result of direct trauma and later of pressure altered static conditions and reduced functional activity. A number of factors were thus responsible simultaneously for the changes.

2 In all other instances the changes were less marked. No changes at all ensued following only ligation of the deep femoral artery and the obturator artery. The regenerative ability of the bone tissue was distinctly evident particularly upon nearly total osteotomy of the neck of the femur.

3 Aseptic necrosis of the bone could be demonstrated by histopathologic autoradiographic (using radio-phosphorus) microradiographic and roentgenologic methods. The best indications were found to be the degenerative changes in the nucleus of the osteocyte and the karyolysis of this cell although the occurrence of granulation tissue even before this stage or emptiness of the cancellous spaces might be considered to be signs of incipient necrosis of the bone.

4 The practice of speaking of avascular necrosis of the bone is inconsistent with the present results. Aseptic necrosis of the bone is suggested as a preferable term for the phenomenon on the grounds that it better implies the presence of numerous factors conducive to such necrosis.

## REFERENCES

- (Abbreviations according to »International Code for the Abbreviation of Titles of Periodicals» in World Medical Periodicals 2nd ed World Medical Association New York 1957)
- ARDEN G P Radioactive isotopes in fractures of neck of femur *J Bone Jt Surg* 1960 42 B 21—27
- ARDEN G P and VBALL N Use of radioactive phosphorus in early detection of avascular necrosis in femoral head in fractured neck of femur *Proc roy Soc Med* 1953 46 344—346
- ARMSTRONG J R Traumatic dislocation of hip joint *J Bone Jt Surg* 1948 30 B 430—445
- AUBIGNE R M d Traitement des nécroses traumatiques de la tête du fémur Wiederherstellungsschrit u Traum 1953 1 108—121
- AUBIGNE R M d and CORMIER Necrose traumatique de la tête du fémur en dehors des pseudarthroses expose *Rev Chir orthop* 1956 42 246—264
- AUBIGNE R M d and HERTZOG P Déformations de la tête et du col du fémur apres enclouage pour fracture. *J Chir (Paris)* 1943 59 89—100
- AXHAUSEN G Knochennekrose und Sequesterbildung *Dtsch med Wschr* 1914 11 111—115
- AXHAUSEN G Die Nekrose des proximalen Bruchstücks beim Schenckelhalsbruch und ihre Bedeutung für das Hüftgelenk *Arch klin Chir* 1922 120 325—346
- AXHAUSEN G Die aseptische Knochennekrose und ihre Bedeutung für die Knochen- und Gelenkchirurgie *Acta chir scand* 1926 40 369—396
- AXHAUSEN G Über anatomische Infarkte am Knochenstern und ihre Bedeutung für die Lehre von den primären Epiphyse nekrosen *Arch klin Chir* 1928 151 72—98
- AXHAUSEN G and BERGMANN E Die Ernährungsunterbrechungen am Knochen In *Handbuch der speziellen pathologischen Anatomie und Histologie* IX, 3 Lds F Henke and O Lubarsch Springer Berlin 1937 pp 118—203
- BADO J L Experiences in treatment of intracapsular fractures of neck of femur *J Bone Jt Surg* 1948 30-A 294—302
- BANCROFT F W Bone repair following injury and infection *Arch Surg (Chicago)* 1922 5 646—677
- BANCROFT F W and MURRAY C R (Eds) *Surgical Treatment of Motor Skeletal System* II Lippincott, Philadelphia 1945
- BANKS S W Aseptic necrosis of femoral head following traumatic dislocation of hip report of nine cases *J Bone Jt Surg* 1941 23 753—781
- BARCLAY A F Micro-Arteriography and Other Radiological Techniques Employed in Biological Research Blackwell Oxford 1951
- BARTH A Ueber histologische Befunde nach Knochenimplantationen *Arch klin Chir* 1893 46 409—417
- BENASSI E Lo sviluppo e il fismo dello scheletro degli arti in rapporto alla allacciatura dei vasi principali *Arch ital chir* 1931 28 49—68

- BENASSY J and LAMARE, J P., Role de la thrombose veineuse dans la pathogenie de la coxarthrose Rev. Chir. orthop. 1956 42 337—342.
- BENNETT G A, WAINE, H and BAUER W. Changes in knee joint at various ages with particular reference to nature and development of degenerative joint disease. Commonwealth Fund New York 1942.
- BENTZON P G K., New theory about pathogenesis of coxa plana and other manifestations of »local dyschondroplasia« Brit. J. Radiol. 1926 31 439—440
- BERGMANN E., Theoretisches, klinisches und Experimentelles zur Frage der aseptischen Knochennekrosen. Dtsch. Z. Chir. 1927 206 12—87
- BLOUNT W P. Don't throw away cane. J. Bone Jt Surg. 1956 38 A 695—708
- BOBECIAO W P and HARRIS, W R., Radiographic density of avascular bone. J. Bone Jt Surg. 1960 42 B 626—632.
- BOHLER, J., Die sogenannten Schenkelkopfnnekrosen nach traumatischen Hüftverrenkungen. Wiederherstellungschir. u. Traum. 1957 4 75—108
- BOHLER, L. and ENDER, J., Hüftkopfnnekrosen nach der Schenkelhalsnagelung ihre Häufigkeit und Versuche der Verhütung. Wiederherstellungschir. u. Traum. 1953 1 122—146
- BONFIGLIO M., Aseptic necrosis of femoral head in dogs. Surg. Gynec. Obstet. 1954 98 591—599
- BONFIGLIO M., and BARDENSTEIN M B., Treatment by bone grafting of aseptic necrosis of femoral head and non union of femoral neck (Phemister technique) J. Bone Jt Surg. 1958 40-A 1329—1346
- BONN., Experimentelle Untersuchungen zur Frage der knöchernen Heilungsfähigkeit medialer Schenkelhalsfrakturen. Zbl. Chir. 1923 50 1454
- BOYD G A., Autoradiography in Biology and Medicine. Acad. Press New York 1955
- BOYD H B and GEORGE, I L., Complications of fractures of neck of femur. J. Bone Jt Surg. 1947 29 13—18
- BOYD H B and GEORGE, I L., Fractures of hip: results following treatment. J. Amer. med. Ass. 1948 137 1196—1199
- BOYD H B, ZILVERSMIT D B and CALANDRUCIO R. A., Use of radio-active phosphorus ( $P^{32}$ ) to determine viability of head of femur. J. Bone Jt Surg. 1955 37 A 260—269 276
- BOYD H. H., Avascular necrosis of head of femur. In American Academy of Orthopaedic Surgeons Instructional Course Lectures 14. Edwards Ann Arbor 1957 pp 196—204
- BRODETTI A., Blood supply of femoral neck and head in relation to damaging effects of nails and screws. J. Bone Jt Surg. 1960 42 B 794—801
- BROWER, T D, AKAHOSHI Y and ORLIC, P., Diffusion of dyes through articular cartilage in vivo. J. Bone Jt Surg. 1962 44 A 456—463
- BRENSCHWIG A., Experimental infarction of bone marrow. Proc. Soc. exp. Biol. (N. Y.) 1929—30 2: 1040—1051
- BLACKHARDT E., Perthes Osteochondritis dissecans und Coxa vara infantum im Tierexperiment. Helv. chir. Acta 1948 15 3—23
- BLARROWS H. J., Coxa plana with special reference to its pathology and kinship. Brit. J. Surg. 1941—42 29 23—36
- CAMPBELL, W C., Campbell's Operative Orthopaedics I. Eds J S Speed and Robert A Knight. Kimpton, London 1956
- CARLSON H., Reactions of rabbit patellar cartilage following operative defects: morphological and autoradiographic study. Acta orthop. scand. 1957 Suppl. 28

- CARRELL, B. and CARRELL, W. B. Fractures in neck of femur in children with particular reference to aseptic necrosis. *J Bone Jt Surg* 1941 23 225—239
- CELLA, C. Note anatomico-funzionale sul legamento rotondo del femore. *Chir Organi Mov* 1934 19 207—213
- CHANDLER, S. B. and KRELSCHER, P. H. Study of blood supply of ligamentum teres and its relation to circulation of head of femur. *J Bone Jt Surg* 1932 14 834—846
- CHENEY, J. Recherches sur la physio-pathologie de la hanche vascularisation de la tete et du col femoral. *Rev Orthop* 1947 33 7—23
- CHENEY, J. Introduction à la pratique chirurgicale de la hanche recherches anatomiques personnelles deduction chirurgicales pratiques. Douin Paris 1954
- CHRISTOPHE, K., HOWARD, L. G., PORTER, T. A. and DRISCOLL, A. J. Study of one hundred and four consecutive cases of fractures of hip. *J Bone Jt Surg* 1953 35 A 729—735
- CLAFFEY, T. J. Avascular necrosis of femoral head. *J Bone Jt Surg* 1960 42 B 802—809
- COLOMAN, P. C. and JONES, E. D. Aetioembolism of bone marrow experimental study. *Arch Surg (Chicago)* 1948 56 161—171
- COMPÈRE, E. L. and WALLACE, G. Etiology of aseptic necrosis of head of femur after trans-cervical fracture. *J Bone Jt Surg* 1942 24 831—841
- COOPER, A. Treatise on Dislocations and on Fractures of Joints. 2nd ed. The Author London 1823
- CORBIN, K. B. Alterations in hip joint after dearterialization. *Arch Surg (Chicago)* 1937 35 1145—1158
- CORNIL, V. and COURAT, D. P. Du col Au point de vue experimental et histologique. *J Anat (Paris)* 1904 40 113—179
- DE HAAS, W. and MACNAB, J. Fractures of neck of femur method of assessing viability of femoral head. *S Afr med J* 1956 50 1005—1010
- DEBOIS, M. Die Hüftkopfnekrose nach der Schenkelhalsnagelung ihre Häufigkeit und Versuche zur Verhütung Wiederherstellungschir u Trauma 1953 1 96—107
- ELSHOLM, R. and NORBACK, B. On relationship between articular changes and function. *Acta orthop scand.* 1951 21 81—98
- LEOESSER, L. On nature of neuropathic affections of joints. *Ann Surg* 1917 66 201—207
- ENGSTROM, A. Histo-radiography in Analytical Cytology. Ed by Robert C. Mellors. Blakiston New York 1955
- ETIENNE, and GRANEL. A propos de la circulation du ligament rond. *Rev Orthop* 1949 35 538
- EVRE BROOK, A. L. and PRIDIE, K. H. Intracapsular fractures of neck of femur final results of 75 consecutive cases treated by closed method of pinning. *Brit. J Surg* 1941—42, 29 115—138
- FITZGERALD, P. J. and ENGSTROM, A. Use of ultraviolet microscopy roentgen ray absorption and radiavotographic techniques in study of neoplastic disease. *Cancer* 1952, 5 643—677
- GALLIE, W. E. Avascular necrosis involving articular surfaces. *J Bone Jt Surg* 1956 38 A 732—738
- GRAH, M. R. A. Experimental considerations in Perthes's disease. *Med. J Aust* 1930—1 207—209
- HABERMAIER, H. Ein Fall einer seltenen Ernährungsstörung des Oberschenkelkopfes nach traumatischer Hüftverrenkung. *Fortschr Röntgenstr* 1955 82 414—416
- HALDTELL, O. Die aseptischen Chondro-osteonekrosen. Walter de Gruyter Berlin 1954
- HALDEMAN, K. O. Healing of joint fractures clinical and experimental study. *J Bone Jt Surg* 1938 20 912—922

- McMANUS J F A Histological and histochemical uses of periodic acid *Stain Technol* 1948 23 99—108
- MARCY G H and FLETCHER R S Modification of posterolateral approach to hip for insertion of femoral head prosthesis *J Bone Jt Surg* 1954 36-A 142—143
- MILTNER, L J and HU C H Osteochondritis of head of femur experimental study *Arch Surg (Chicago)* 1933 27 645—657
- MOVAT H Z Demonstration of all connective tissue elements in single section *Arch Path. (Chicago)* 1955 60 289—295
- MÜLLER W Experimentelle Untersuchungen über Nekrosen und Umbauprozesse am Schenkelkopf nach traumatischen Epiphysenlosungen und Luxationen und ihre klinische Bedeutung *Bruns Beitr klin. Chir* 1924 137 490—522
- MÜLLER, W Über das Verhalten des Knochengewebes bei herabgesetzter Zirkulation und das Bild von Nekrose der Zwischenlamellen *Bruns Beitr klin Chir* 1927 138 614—624
- MUSSBICHLER H Arterial supply to head of femur arteriographic study in vivo of lesions attending fracture of femoral neck *Acta radiol (Stockh)* 1956 46 533—546
- NAGURA S Das Wesen und die Entstehung der Osteochondritis dissecans Königs (bzw der Perthes Kohler II und der ähnlichen Krankheiten und Veränderungen an wachsenden Knochenenden) *Zbl Chir* 1937 64 2049—2059
- NAGURA S Ein weiterer Beitrag zur Entstehung der Pertheschen Krankheit *Zbl Chir* 1938 65 1707—1712.
- NAGURA S and KOSLGE S Die Pathogenese und das Wesen der Pertheschen Krankheit experimentelle Beiträge zur Entstehung der Pertheschen Krankheit *Arch klin Chir* 1938 191 347—371
- NORDBY L J Evaluation of aseptic necrosis *Spectator* January 1960
- NORDENSON N G Sur la vascularisation de la tête du femur par la voie du ligament rond femoral. *Lyon chir* 1938 33 178—187
- NUSSBAUM A Über Osteochondritis coxae juvenilis Calve Legg Perthes *Dtsch med Wschr* 1923 49 849—850
- NUSSBAUM Demonstration über die Erzeugung von Osteochondritis juvenilis auf experimentellem Wege *Zbl Chir* 1923 50 937
- NUSSBAUM A Die arteriellen Gefässe der Epiphysen des Oberschenkels und ihre Beziehungen zu normalen und pathologischen Vorgängen *Bruns Beitr klin Chir* 1924 130 492—535
- NUSSBAUM A. Die Gefässe am oberen Femurende und ihre Beziehungen zu pathologischen Prozessen II Mitteilung *Bruns Beitr klin. Chir* 1926 137 332—345
- NASTROM G Further experiences with osteosynthesis of medial fractures of femoral neck with aid of three nails («multiple nailing») with special reference to indications for reconstructive operations on hip-joint *Acta chir scand* 1954 107 89—112
- OLIVSON TH Beitrag zur Kenntnis der Verbindungen zwischen A Ilica Interna und A Femoralis beim Menschen nebst tierexperimentellen Studien über die Morphologie des Kollateralkreislaufs nach Unterbindung der A Ilica Externa und A Femoralis *Acta chir scand* 1942, 86 Suppl 67
- PALLETTA J B Exercitationes pathologicae Mediolani 1820
- PAULS B Traumatic dislocations of hip late results in 76 cases *Acta orthop scand* 1951 21 99—112.
- PEARSE, U E Experimental study of arterial collateral circulation. *Ann Surg* 1928 88 227—232



- B., Endausgänge von traumatischen Hüftgelenkverrenkungen Arch orthop Unfall  
Chir 1939 39 135—143
- EMISTER D B Radium necrosis of bone. Amer J Roentgenol 1926 16 340—348
- EMISTER, D B Repair of bone in presence of aseptic necrosis resulting from fractures  
transplantations and vascular obstruction. J Bone Jt Surg 1930 12 769—87
- EMISTER D B Aseptische Knochennekrose bei Frakturen Transplantationen und Gefäß  
verschlüssen. Z. orthop Chir 1931 35 161—186
- EMISTER, D B Fractures of neck of femur dislocations of hip and obscure vascular dis  
turbances producing aseptic necrosis of head of femur Surg Gynec. Obstet 1934 59  
415—440
- EMISTER D B Bone growth and repair Ann Surg 1935 102 261—285
- EMISTER, D B Pathology of ununited fractures of neck of femur with special reference to  
head. J Bone Jt Surg 1939 21 681—693
- FRITS F N and ORLETZ B E Aseptic necrosis of head of femur following traumatic dis  
location. J Bone Jt Surg 1939 21 101—110
- FRILANDER P V Costa vera infantum Acta orthop scand 1960 Suppl. 48
- FRIGALLO J Histochemical studies on vital and post mortem skin wounds experimental  
investigation on medicolegally significant vital reactions in early phase of wound healing  
Ann. Med. exp. Tenn 1961 39 Suppl. 6
- FRISSEN P M Surgical anatomy and pathology of supraspinous and interspinous ligaments  
of lumbar spine with special reference to ligament ruptures Acta orthop scand 1960  
Suppl. 46
- FRONZ, F W Arteriography of hip joint for predicting and results in intracapsular and inter  
trochanteric fractures of femur Amer J Surg 1953 86 404—409
- FRUSSE, O Frattura del collo del femore e necrosi dell'epifisi Arch. Patti Chir. Organi Mov  
1952 2 33—39
- FRUTISHAUSER E Vascularity of bone in relation to pathological studies (Paget's disease, Sudeck's  
atrophy, osteoarthritis of hip) In Ciba Foundation Symposium on Bone Structure and  
Metabolism Eds G E W Wolstenholme and Cecilia M O'Connor Churchill Lon  
don, 1956 pp 239—248
- FRUTISHAUSER E and MAJNO G Les lésions osseuses par surcharge dans le squelette normal  
résultats expérimentaux Schweiz med Wschr 1949 9 281—288
- FRUTISHAUSER F and MAJNO G Physiopathology of bone tissue: the osteocytes and funda  
mental substance Bull. Hosp. Jt Dis (N.Y.) 1951 12 468—490
- SACERDOTE G Sulle conseguenze di interruzioni circolatorie e di fratture nelle epifisi di ani  
mali in accrescimento Chir. Organi Mov 1931 16 291—305
- SANTOS J V Changes in head of femur after complete intracapsular fracture of neck their  
bearing on nonunion and treatment Arch Surg (Chicago) 1930 21 470—530
- SARPEL P H Traité d'anatomie descriptive II Delahaye Paris 1809
- SCAGLIETTI O Über die Nekrose der Femurepiphyse bei Schenkelhalsfrakturen Wundheilung  
u. Traumat. 1953 1 147—161
- SCHWEIGERY M Das Ligamentum teres femoris und seine Gefässe pathologisch anatomische  
Untersuchungen zur Frage der Bedeutung der Ligamentgefässe für die Ernährung des  
Oberschenkelkopfes Z. Orthop 1936 65 297—317
- SHERMAN M S and SELAKOVICH W G Bone changes in chronic circulatory insufficiency  
histopathological study J Bone Jt Surg 1957 39 A 892—901
- SMITH W S BRETON R J and COLEMAN C R Sequelae of experimental dislocation of  
weight bearing ball — and — socket joint in young growing animal gross alterations  
in bone and cartilage J Bone Jt Surg 1958 40-A 1121—1127

- SMITH PETERSEN M. N., Approach to and exposure of hip joint for mold arthroplasty. *J. Bone Jt Surg* 1949 *31 A* 40—46
- STEEDMAN H. F., Alcian blue 8GS new stain for mucin. *Quart. J. micr. Sci.* 1950 *91* 477—479
- STEVENS J., Value of radioactive isotopes in determining viability of bone. *J. Bone Jt Surg.* 1960 *42 B* 650
- STEWART M. J., and MILFORD L. W. Fracture-dislocation of hip end result study. *J. Bone Jt Surg* 1954 *36 A* 315—342.
- STEWART W. J. Aseptic necrosis of head of femur following traumatic dislocation of hip joint case report and experimental studies. *J. Bone Jt Surg* 1933 *15* 413—438
- TANAKA K. R., CLIFFORD G. O. and AXELROD A. R., Sickle cell anemia (homozygous S) with aseptic necrosis of femoral head. *Blood* 1956 *11* 998—1008
- THOMSEN (Calc. Perthes like changes in head of humerus in swine) Discussion to Robert Hanson's article Epiphyseal necrosis of caput femoris. *Acta orthop scand.* 1939 *10* 331—337
- THOMPSON A. P. and EPSTEIN H. C., Traumatic dislocation of hip survey of two hundred and four cases covering period of twenty-one years. *J. Bone Jt Surg.* 1951 *33 A* 746—778
- (TOVEY, E. B.) Fractures of neck of femur in dogs Unpubl. paper read at Annual Meeting of American Orthopaedic Association 1953 Cited by GALLIE in *J. Bone Jt Surg* 1956 *38 A* 732—738
- TROJAN E., and PERSCHL, A. Die Behandlungsergebnisse von 79 frischen traumatischen Hüftgelenksverrenkungen (HV) und Hüftgelenksverrenkungsbrüchen (HVB) *Ergebn. Chir. Orthop* 1956 *20* 90—169
- TRUETA J. Regeneration of bone and cartilage. *Societe Internationale de Chirurgie. IV Congres, Lisbonne* 1953 Publ. par L. Dejardin. Bruxelles 1954 pp 564—574
- TRUETA J. Normal vascular anatomy of human femoral head during growth. *J. Bone Jt Surg* 1957 *39 B* 358—394
- TRUETA, J. and HARRISON M. H. M., Normal vascular anatomy of femoral head in adult man. *J. Bone Jt Surg.* 1953 *35 B* 442—461
- TUCKER, F. R., Arterial supply to femoral head and its clinical importance. *J. Bone Jt Surg.* 1949 *31 B* 82—93
- TUCKER, F. R., Use of radioactive phosphorus in diagnosis of avascular necrosis of femoral head. *J. Bone Jt Surg.* 1950 *32 B* 100—107
- URIST M. R., Injuries to hip joint. Traumatic dislocations incurred chiefly in jeep accidents in world war II. *Amer. J. Surg* 1947 *74* 586—597
- URIST M. R., Fracture — dislocation of hip joint nature of traumatic lesion treatment, late complications, and end results. *J. Bone Jt Surg.* 1958 *30 A* 679—727
- VEREBY K., Die Blutversorgung des Femurkopfes. *Anat. Anz.* 1942 *93* 225—240
- VOELKMAN R., Mündelung aus der chirurgischen Casusistik und kleinere Mittheilungen. *Arch. klin. Chir* 1864 *3* 330—335
- WALDENSTROM H., Necrosis of femoral epiphysis owing to insufficient nutrition from ligamentum teres clinical study mainly based on experiences of treatment of epiphyseolysis capitis femoris. *Acta chir. scand.* 1934 *75* 185—197
- WALLER, A., Dorsal acetabular fractures of hip (dashboard fractures) *Acta chir. scand.* 1955 *Suppl. 200*
- WALMSLEY T., Epiphysis of head of femur. *J. Anat. (Lond.)* 1914—15 *49* 434—440
- WATSON-JONES, R., Avascular necrosis. Discussion at Meeting of Section of Radiography of Royal Society of Medicine. *Brit. med. J.* 1942 *1* 503—504

- WATSON JONES R. and ROBERTS R. E. Calcification, decalcification and ossification. *Brit. J. Surg.* 1934 21 461—499
- WEIS J. and MÜLLER, H. Die Verhütung der Oberschenkelkopfnnekrosen nach Schenkelhalsbrüchen. *Maschr. Unfallheilk.* 1954 57 18—27
- WISE, R. A. Histological study of transcervical fracture of femur after internal fixation. *J. Bone Jt. Surg.* 1941 23 1 941—947
- WOLCOTT W. E. Circulation of head and neck of femur in relation to nonunion in fractures of femoral neck. *J. Amer. med. Ass.* 1933 100 27—34
- WOLCOTT W. E. Evolution of circulation in developing femoral head and neck. *Surg. Gynec. Obstet.* 1943 77 61—68
- WOLLENBERG A. Über Knochennekrose. *Z. orthop. Chir.* 1928 50 415—431
- ZEMANSKY A. P. Jr. and LIPPMAHN R. H. Importance of vessels in round ligament to head of femur during period of growth, and their possible relationship to Perthes disease. *Surg. Gynec. Obstet.* 1929 48 461—469







ACTA ORTHOPAEDICA SCANDINAVICA  
SUPPLEMENTUM No. 59

---

FROM THE ORTHOPAEDIC HOSPITAL OF THE INVALID FOUNDATION,  
HELSINKI, FINLAND. HEAD- A. LANGE-SKIÖLD, M.D.

THE PATHOGENESIS  
OF EXPERIMENTAL PROGRESSIVE  
SCOLIOSIS

BY

A LANGE-SKIÖLD

AND

J E MICHELSSON

5 APR 1967

A handwritten signature, possibly 'M. Ki', with a large checkmark drawn over it.

MUNKSGAARD

Copenhagen 1962





THE PATHOGENESIS  
OF EXPERIMENTAL PROGRESSIVE  
SCOLIOSIS



FROM THE ORTHOPAEDIC HOSPITAL OF THE INVALID FOUNDATION  
HELSINKI, FINLAND HEAD A. LANGENSKIÖLD MD

THE PATHOGENESIS  
OF EXPERIMENTAL PROGRESSIVE  
SCOLIOSIS

BY

A. LANGENSKIÖLD

AND

J.-E. MICHELSSON



PRINTED IN FINLAND BY TILGMANN  
HELSINKI 196

In an article published in 1961 we reported that progressive scoliosis can always be provoked in rabbits by resecting the posterior end of a rib. Progression up to 170 degrees was observed (Fig. 1). Of 13 different operations on nerve, muscle, and bones in the vicinity of the pinned rib resection was the only one after which scoliosis occurred in every animal.

The systematic search for different experimental measures provoking scoliosis was continued. After section of the ligaments around the proximal ends of 4—5 ribs leaving the ligamenta tuberculi intact scoliosis of 40—115 degrees developed in 10 of 21 animals. Section of all ligaments around the costovertebral joint. 11 animals gave a similar result.

When we studied the scoliotic vertebrae of pigs which had undergone rib resection we observed that the deformity of the arch seemed greater than that of the vertebral body (Fig. 2). This has formerly been found to be true also in scoliotic human vertebrae (Roux). The possible significance of the lamina in the pathogenesis of scoliosis has been stressed by Sommerilli.

Working in our laboratory on problems concerning the sequelae of lumbar resection, Trueta found that structural scoliosis often developed in rabbits after hemilaminectomy of lumbar vertebrae. As the 24th type of operation in our series we performed hemilaminectomy on the right side of 3 thoracic vertebrae in rabbits aged 26—77 days. The hemilaminectomy included removal of the articular processes on the right side. Of the 6 animals 3 survived the operation. A scoliosis of 40—180 degrees appeared in 2 animals. In 11 animals the scoliosis progressed to 80 degrees or more (Fig. 3). As the 1st type after rib resection the scoliosis arising after hemilaminectomy is convex to the side of the operation.

Apparently resection of the posterior ends of ribs and hemilaminectomy are two entirely different operations. Even the approach to the resected parts of the skeleton is different in the 2 operations. However the fact that they may both cause extreme scoliosis led us to search for a common factor in the 2 operations.

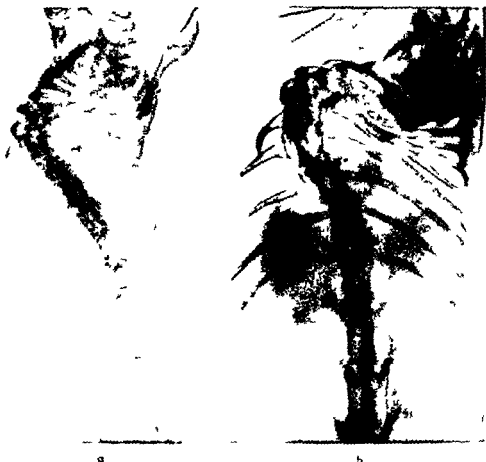


Fig. 1. *Seh* rabbit after excision of the posterior half of the fifth rib: a) 14 days after operation; b) 37 days after operation. At 71 months *Seh* 170 hours.

A study of the anatomy of the human spine shows that there is a ligament connecting the lamina of each thoracic vertebra with the rib (called  $L_{pr}$ ). In the rabbit and the pig there is a corresponding structure. Resection of the posterior half of a rib or hemilaminectomy causes loss of function of the ligament  $L_{pr}$  to show the pain of a rabbit with a colic of Soderberg which was provoked by section of the posterior costal transverse ligament at five levels. By Fick called *hamamentum collicolicum superius ad os sterni*. After exposure of the ligament does not produce colic it appears that the difficulty had arisen as a result of the section of five transverse ligaments.



1. a) Radiograph of tooth (axial view) b) Vertebral c) Axial  
 from Radiograph of b) and lamina taken at a distance from the  
 portion





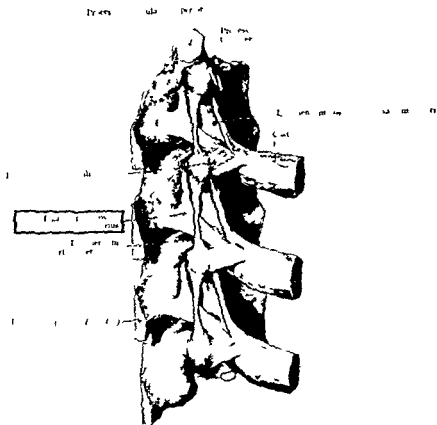


Fig. 4. Ligament connecting the rib to the spine (From Spalteholz)

Severe progressive scoliosis was only seen in a few of 47 rabbits on which section of the posterior and anterior costo-transverse ligaments was done at five levels. Scar formation seemed often to prevent the deforming effect of the operation. There are, however, other facts indicating the importance of the posterior costo-transverse ligament to the stability of the spine.

### Functional scoliosis arising immediately after operation on the spine or in its vicinity

In 66 rabbits in which the posterior ends of five ribs were resected the initial scoliosis was estimated from radiographs taken immediately after the



Fig. 1. Rat tail. *a*) Distribution of myxoma on the right side of lumbar vertebrae I-IV at the operation; *b*) Tail immediately after operation; *b*) Animal killed 13<sup>1</sup>/<sub>2</sub> months later. Scale 1:10 (1 cm).



Fig. Rabbit Mucl<sub>1</sub> connecting laminae and transverse processes of lumbar vertebrae.  
1—V. It existed at the age of 30 days. Stained 1:30 degrees.

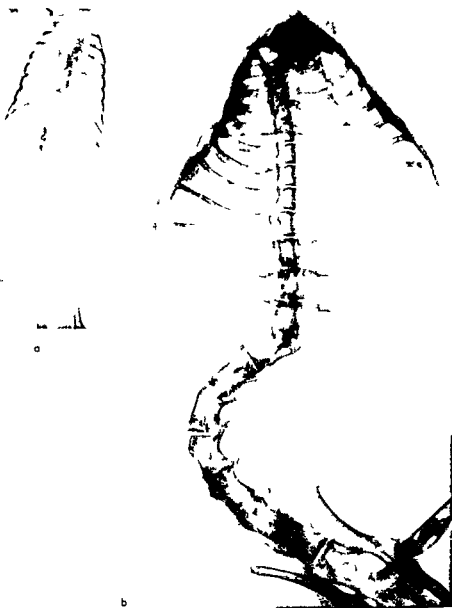
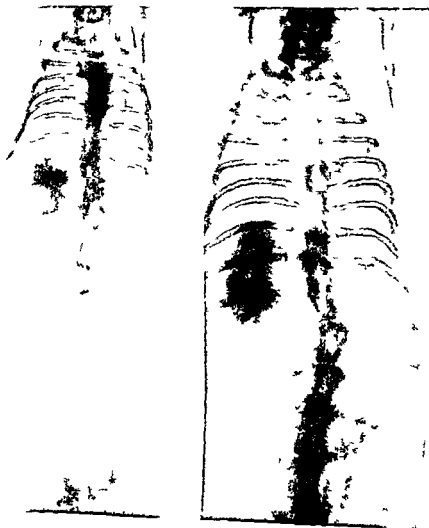


Fig. 1. Hemulammectomy in the right side of lumbar vertebrae I-IV at the top of S. I. (a) Lateral view of the lumbar vertebrae after vertebrae I-IV are removed. (b) Lateral view of the lumbar vertebrae after vertebrae I-IV are removed. Scale bar = 10 mm.

ligaments mentioned above were dissected on the concave side and in some of them a reconstruction with nylon thread of the posterior costotransverse ligament on the convex side was attempted. In some of the rabbits parts of the intercostal muscles were dissected.

In 8 of the 11 animals the spine grew almost straight from a scoliosis of 45-55 degree.

Fig. 8. Rabbit. Hemilaminectomy of thoracic vertebrae VI-X on the right side at the age of 3 days. a) Age 46 days. Scoliosis 9 degrees in resting position. b) Age 46 days. Fixation scoliosis degree when trunk was bent to the right. c) Spine almost straight in resting position 14 days after resection of anterior and posterior costotransverse ligament and part of the intercostal muscle in the interspace Th VI-IX on the convex side. Age 58 days. d) Scoliosis converted to other side. Age 6 months.



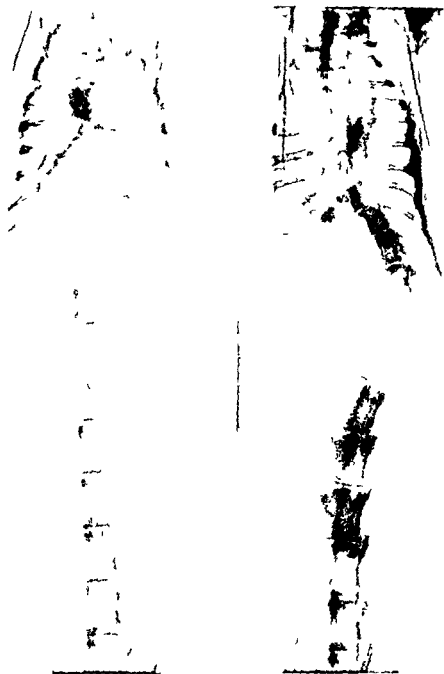


Fig. 9. Radiat. Extortion of the posterior end of the right sixth to tenth rib at the age of 11 yrs. a) Age 6 days. Scoliosis degree. b) Four day after hemilaminectomy of sixth rib. H.V. X. ninth concave side. Note exaggeration of the capital-concave curve.



Fig. 10. 1) Excision of the tumor and of the right sixth to tenth rib at the age of 3 years. Scoliosis 30 degrees four months after operation (The postoperative result).





Fig. 8 shows an example of the straightening effect of a secondary operation of the concave side. Although some spontaneous straightening has been seen in scoliotic rabbits, there is no doubt that the procedure on the concave side can neutralize the deforming forces.

Some straightening effect was also seen after hemilaminectomy on the concave side of curvatures provoked by rib resection. Fig. 9 demonstrates a phenomenon which sometimes appeared after secondary operation. A hemilaminectomy on the same level on which the initial provoking rib resection had been made on the opposite side caused progression of the caudal secondary curve. This experiment shows clearly the growth conducting power retained by both operations and the importance of a proper localization of any curative operation. In addition it may indicate a potential danger if untimely use is made of these methods in the treatment of scoliosis in man.

### Experimentally provoked scoliosis in pigs

Resection of the posterior ends of five ribs was carried out on 12 pigs (Fig. 10). In 11 of these a scoliosis of 20—75 degrees developed (average 37 degrees).

In 10 pigs the posterior and the anterior costo transverse ligaments were cut at 3 levels. A scoliosis of 30—45 degrees developed in 9 of these animals. In 4 of the pigs the ligaments mentioned and the intercostal muscles at a distance of some centimeters from the vertebrae were discised on the concave side 3—4 weeks after the first operation. In two of the four animals a reconstruction with nylon thread of the discised posterior costo transverse ligaments on the convex side was also attempted. After these secondary operation the scoliosis in three of the pigs was corrected to less than 10 degrees (Fig. 11). In the 3 scoliotic animals which had no secondary operation there remained a deformity of at least 24 degrees.

Fig. 12 shows the deformity of the ribs and the rotation of a vertebra in a pig made scoliotic by a soft tissue operation.

Scoliosis exceeding 90 degrees has until now been seen in pigs only after progression following a second operation (Fig. 13).

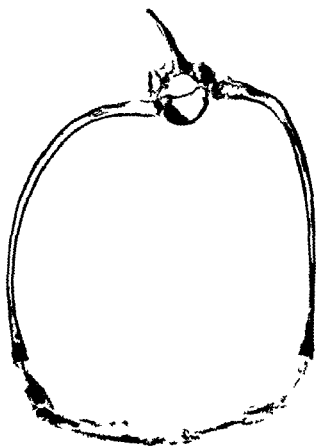


Fig. 11. Vertebra and rib at the apex of a scolioz (30 degree) provoked by section of the posterior and anterior costal transverse ligament and parts of the intercostal muscle at six levels. Note rotation of vertebra and distortion of the thoracic cage



Fig. 13.11. Excision of the posterior end of the seventh to eleventh rib on the right side at the age of one month. Larunectomy of vertebrae Th VI—XI 40 days later when a scoliosis of 40 degree had developed. At the age of 7 months scoliosis over 100 degree.

## Discussion

The only factor which the scoliosis provoking operations seem to have in common is the loss of function of the posterior costo-transverse ligament (*ligamentum costotransversarium ad medium*). In textbooks on human anatomy it has been pointed out that this ligament transfers the effect of the rhomboids and the longissimus muscles from the ribs to the vertebrae (BRYAN).

We have arrived at the conclusion that the loss of function of the posterior costo-transverse ligament is the most important factor provoking scoliosis in the experiments. The most effective suppression of this function is the resection of a part of the skeleton to which the ligament is attached (resection of the end of the rib or hemithoractomy).

The deforming effect of unilateral division of the posterior costo-transverse ligament and the straightening effect of its section on the concave side of a provoked scoliosis at once seem to justify the following conclusions: as to the pathogenesis of the spinal deformities described above.

*The posterior costo-transverse ligament in transmitting the effect of normal muscle tone to the spine is of decisive importance to its equilibrium and symmetrical growth.*

*A unilateral protracted insufficiency of the function of the posterior costo-transverse ligament at several levels of the spine of a growing rabbit or pig causes progressive scoliosis of the same type that is seen in man.*

In the experiments described above the deforming forces have been transmitted to the vertebral body by the arch.

Loss of function of the posterior costo-transverse ligament on one side is followed by shortening of the corresponding ligament on the other side. This contracture is soon followed by contracture of other structures.

*Scar formation in the region of the posterior costo-transverse ligament or shrinkage of this structure may affect the growth of the vertebra and cause scoliosis. Excision may reestablish the function of a diseased ligament.*

It is obvious from the anatomy of the posterior costo-transverse ligament that shrinkage of it leads to a deformity composed of lateral flexion and rotation of the spine.

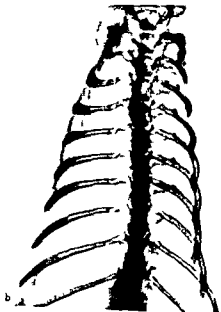
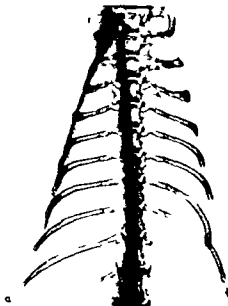
Some facts pointing to connections between the experimental results and scoliosis in man may be stressed.

Fig. 11 shows a scoliosis appearing as a sequel of poliomyelitis in a child. On the convex side there is typical drooping of the ribs. Fig. 12 shows a radiograph of the spine and parts of the ribs of a normal rabbit. All material had been excised from the left half of the specimen which had been

FIG 14 Human Section after  
 fibrinolytic Note drooping of rib on  
 the convex side →



FIG 15 Rabbit a) Thoracic spine and  
 posterior halves of the ribs of a normal  
 animal. All muscles have been cut  
 away from the left half of the specimen.  
 Radiograph taken with specimen in an  
 erect position b) Same specimen as a)  
 On the left side the posterior  
 costal transverse ligament have been  
 dissected. The ribs have developed  
 a large p. Compare with fig 14. 4x



radio-graphed in an erect position Fig. 10a shows the same specimen after division of the posterior costo transverse ligaments on the left side. The ribs are drooping. Insufficiency of the function of this ligament may perhaps be a factor through which paralysis affects the equilibrium and growth of the pine.

Fig. 10b shows a radio-graph of the pine of a girl aged 10 years. In early childhood an abscess had caused a scar in the tissues on the left side of the pine. The scar extended to the vertebral laminae. The picture shows the pine one week after excision of the scar. There is scoliosis of 30 degrees and considerable structural change. Fig. 10b shows the same spine one year later. There was no other treatment than the operation. The spine had straightened almost 10 degrees. In connection with the excision of the scar performed by A. L. VASSERSTEDT the medial parts of several posterior costo transverse ligaments had probably been removed. At the time of the operation we did not yet know the importance of the ligaments in the pathogenesis of experimental scoliosis. This case proves that a moderate structural change in a child may correct itself by growth after an adequate operation in which the deforming factors are removed or transferred to the other side.

The facts reported in this article constitute the essential results of experiments on about 600 rabbits and 30 pigs.

Many other observations made in the experiment will be reported in a thesis by J. L. MEUNISSEN.

## Summary

Progressive scoliosis can always be produced in rabbits by removing the posterior end of 1—6 ribs (Fig. 1). By hemilaminectomy of five vertebrae in the thoracic spine the same condition arises in more than 2/3 of the experiments (Fig. 3). The only factor common to the two operations seems to be the loss of function of the posterior costo transverse ligament ligamentum alio-costae apicarium ad arcum. Progressive scoliosis can be provoked in rabbits by unilateral section of this ligament at five levels in the pine. The primary curve is convex to the side of the operation.

Moderate structural scoliosis is always seen in pigs after unilateral division of the anterior and the posterior costo transverse ligaments at a level in the pine (Fig. 11). Scoliotic spines in rabbits and pigs can be made to grow straight by section on the convex side of the ligaments mentioned and part of the intercostal muscles (Figs. 8 and 11).

A proof of unilateral insufficiency of the function of the posterior

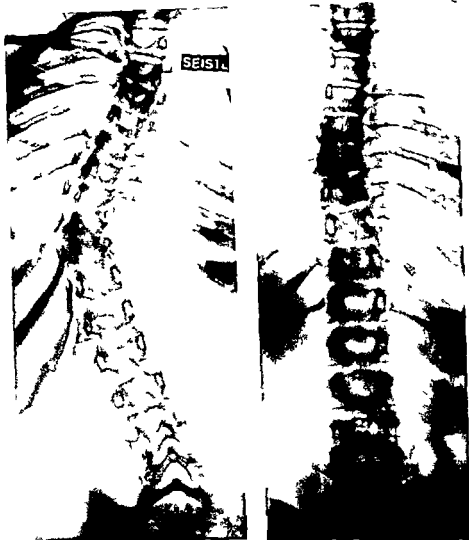


Fig. 10 Human Scoliosis of 10 degrees in a child aged 10. There was a car from an abscess extending to the vertebral laminae. a) Radiograph taken one week after excision of the car. b) Radiograph one year after operation. The spine was almost straight.

co to transverse ligament at several levels of the spine of a growing rabbit or pig causes progressive scoliosis of the same type as is seen in man. This ligament, in transmitting the effect of normal muscle tone to the spine is of decisive importance to its equilibrium and symmetrical growth.

## REFERENCES

- BRATS H. Anatomie des Menschen. I Band. Julius Springer, Berlin 1931  
p. 114
- FICK R. Handbuch der Anatomie und Mechanik der Gelenke. G. Fischer,  
Jena 1931
- LANGENSKIÖLD A. & MICHELSSON J. E. Experimental progressive scoliosis in  
the Rabbit. *The Journal of Bone and Joint Surgery* 43B 116 1961
- MICHELSSON J. E. Unpublished observations
- ROAF R. Rotation Movements of the Spine with special Reference to Scoliosis  
*The Journal of Bone and Joint Surgery* 40B 91 1958
- SOMERVILLE I. W. Rotational Lordosis. The Development of the Single  
Curve. *The Journal of Bone and Joint Surgery* 34B 421 1952
- SPALTENHOLZ W. Handatlas der Anatomie des Menschen. I Band. S. Hirzel,  
Leipzig 1904
- TROLEPP H. Unpublished observations

We are indebted to the Sigrid Juselius Foundation for grants which made this work possible

The operations on pigs were carried out at the School for Agriculture of the Invalid Foundation Saira Minksa. We wish to thank the director, Mr A. Rannio and Mr T. Räsänen for their kind cooperation











FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI, (HEAD PROFESSOR K. E. KALLIO)

# INJURIES OF THE THORACO-LUMBAR SPINE WITH PARAPLEGIA

BY  
ERKKI KALLIO

---

MUNKSGAARD  
COPENHAGEN 1963



ACTA ORTHOPAEDICA SCANDINAVICA

SUPPLEMENTUM No 60

---

FROM THE CLINIC FOR ORTHOPAEDICS AND TRAUMATOLOGY  
UNIVERSITY OF HELSINKI (HEAD PROFESSOR A. E. KALLIO)

# INJURIES OF THE THORACO-LUMBAR SPINE WITH PARAPLEGIA

BY

ERKKI KALLIO

---

MUNKSGAARD  
COPENHAGEN 1963

*Printed in Finland*  
Helsinki 1963  
MARGATORIN KIRJAPAINO



## CONTENTS

### INTRODUCTION

### SURVEY OF THE LITERATURE

- The Type of Injury to the Neural Elements
- Complete or Incomplete Lesion of the Cord?
- How to Distinguish Root Lesions from Cord Damage?
- Stable and Unstable Spinal Injuries
- Operative Indications

### OWN INVESTIGATIONS

- Description of the Material
- Fractures between 1st and 11th Dorsal Vertebrae — Practically Pure Cord Lesions
- Fractures of Dorso Lumbar Junction (D12 and L1) — Mixed Cord and Root Lesions
- Fractures between 2nd and 5th Lumbar Vertebrae — Root Damage Only
- Methods of Treatment and Results

### DISCUSSION AND CONCLUSIONS

### SUMMARY

### REFERENCES



## INTRODUCTION

Until the end of the second world war traumatic paraplegia used to be a classical example of hopeless case. Since then considerable improvement has occurred in the unders anding and treatment of injuries to the spine with paraplegia. The work of GUTSMAN at the Stoke Mandeville Hospital near London is widely known and recognised. Amazing results has been obtained by careful nursing and early rehabilitation of the paralysed patients. HOLDSWORTH and HANBY of Sheffield have made great contributions in the surgical treatment of traumatic paraplegia. They introduced the idea of internal fixation or internal splinting, of the fractures to protect the neural elements capable of recovering. Mc RO and ROCHES have been the pioneers in this field in America.

All authorities agree that the main points in the treatment of paraplegic patients are good nursing, prevention of complications and early rehabilitation. But there seems to be no unity in the actual early management of the cases. What exactly should be done? Some surgeons are in favour of early laminectomy, others advocate open reduction and plating and some are strongly against operative treatment in most cases. Should closed reduction be done and if so how can it be retained? Is plaster jacket useful or harmful? Should plaster bed ever be used in these cases? What is the value of Queckenstedt test in contemplating operation? What is the main purpose of the operation: reduction decompression or protection to prevent further damage? Many questions arise and the answers are not easy to find. The views presented in the relevant literature are still rather controversial and confusing.

The present writer became interested in the problems of traumatic paraplegia while attending a short course in accident surgery in Sheffield in 1960. The writer visited the Spinal Injuries Unit at the Edgemoor Hospital and became inspired by the views presented by HOLDSWORTH and HANBY. Since April 1961 the author has been responsible of the treatment of paraplegia from fractures of the cervicolumbar spine in the Clinic for Ortho-

paedics and Traumatology University of Helsinki. He has followed the policy of the last mentioned British authors. Ten patients have been treated accordingly so far. For comparison the writer collected data from previous cases. He could find 29 cases of traumatic paraplegia of the thoracolumbar spine treated in the same clinic during the previous ten consecutive years. The present study is based apart from the literature to the 36 cases from which the author has enough data to make some conclusions.

Although the present writer's personal experience in the treatment of traumatic paraplegia from fractures of the thoraco-lumbar spine is limited the results obtained have interested the writer for further study.

## SURVEY OF THE LITERATURE

### The Type of Injury to the Neural Elements

Injuries to the spinal cord can be divided into different groups according to the type of the lesion. DAVIDSON divides them as follows: concussion, contusion, laceration, hematorachis, hematomyelia, and compression. FOERSTER (quoted by GUTTMAN) adds to this an ischaemic lesion due to thrombosis in the small blood vessels of the cord. The pathological picture behind neurological symptoms is, according to SCARFF, due to oedema and concussion of the cord, hemorrhage into the cord (hematomyelia), compression of the cord by fractured or misaligned vertebrae without actual transection, transection of the cord by elements of the vertebral column. The lesion is often a combination of different types of damage.

Complete recovery is possible only from concussion. Noticeably recovery from other lesions is dependent on the amount of concussion or oedema which is combined to them. Destruction of the cord is irreparable because the central nervous system has no noteworthy regenerative power. An optimistic attitude towards future is presented by BOSHES, who writes: "In the past few years have come the first glimmerings of hope that the old dream of regeneration of fibers in an injured spinal cord may some day come true."

Concussion of the cord (*commotio medullae spinalis*) is regarded as functional disturbance in the cord due to injury. This is comparable to concussion of the brain. SCARFF describes oedema and concussion under the same heading. Oedema occurs in every spinal injury which is severe enough to produce neurological symptoms. The cord can swell to twice its normal size within a few minutes. Such oedematic cord fills the dural sac even with some pressure underneath it. Oedema is the most important cause of manometric block (positive Queckenstedt test) during the first 18-72 hours after the injury (SCARFF). It begins to subside after the third post-traumatic day and the symptoms produced by it should disappear in a

week. It is not clearly defined in the literature how soon the symptoms and signs due to concussion of the cord should subside but it is understood that this should happen pretty early. When concussion and oedema are combined together they will lengthen the symptoms produced by each of them. SCARFF writes further "In the more severe and prolonged cases it may even be that the two mechanisms just named are augmented by the occurrence of numerous petechial hemorrhages into the grey matter of the cord." According to DAVIDSON the hemorrhages when present are usually due to diapedesis and are rarely caused by rupture of the vessels.

The picture of complete paraplegia in incomplete lesions of the cord is thus partly due to irreparable damage of the cord and partly to changes which subside. The term spinal concussion is used to that part of the cord lesion where neurological recovery is possible regardless of the actual pathology behind it. Although *commotio medullae spinalis* and *oedema of the cord* are different things they are both included in the concept of spinal concussion used by HOLDSWORTH for example.

Spinal concussion and spinal shock have been confused by many authors. These are two quite different conditions which should be clearly separated. Spinal shock is the state of the distal segment of the cord immediately after complete division. The distal segment of the cord is cut off from cerebral centers and has not yet got its automatic "decerebral" function. This is a transient state before the appearance of increased reflex activity. In spinal shock there is no motor activity, the corresponding segments are anesthetic, the paralysis is flaccid and the reflexes are away. In fact the anal and bulbocavernosus reflexes may remain (NAFFZIGER, HOLDSWORTH and HARDY 1953). Spinal shock can disappear fairly quickly or last for many weeks before the next phase of neurological behaviour of the distal spinal segments appear (BOSHES, BUSCH, CHUSID and McDONALD, DAVIDSON, HOLDSWORTH and HARDY, PEELE and others).

When the spinal shock subsides isolated reflex activity begins to occur. The reflex activity and flexor response phenomenon manifesting at this stage have been erroneously indicated as signs of favourable prognosis. It should be clear that no normal sensation or voluntary motor power can follow after spinal shock. HOLDSWORTH states "The return of reflex activity below a cord lesion without recovery of sensation or of normal motor power is therefore never an indication of continuity of the cord axons but on the contrary is a certain sign of irrecoverable cord division".

After the stage of increased reflex activity in complete lesions of the cord comes the terminal phase. The reflexes diminish and the physical state

the general health of the patient deteriorates gradually towards final debility

Contusion of the spinal cord is a severe localised injury which as such is irreparable but which can be partly combined with marked oedema and hemorrhage. In laceration there is also rupture of the membranes with leakage of the cerebrospinal fluid into the surrounding tissues. Infection can ensue and seriously complicate the picture. Hemorrhachis bleeding into the spinal canal can be epi intra or subdural or subarachnoid in nature. If there is marked hemorrhage it can lead to compression and to myelopathic changes. That is very rare (DAVIDSON). Hematomyelia haemorrhage in the cord tends to spread in the gray matter longitudinally. Hematomyelia can be caused by direct or indirect injury. Even violent muscular strain may produce it. Usually the bleeding occurs immediately but it can follow an interval of several hours. Compression acts directly or indirectly by preventing blood supply. Scarring can be responsible for gradual late compression. Posttraumatic thrombosis in the vessels of the cord is an assumed event presented by FOERSBERG (quoted by GURTMANN).

Injuries of the spinal cord and nerve roots are usually due to fractures and fracture dislocations which are clearly to be seen in the radiographs. But it is known that traumatic lesions can occur in the spinal cord without noticeable radiographic changes. It should be possible at least theoretically to differentiate segmental lesions from tract lesions.

In the segmental syndrome sensitivity is abolished or diminished in the affected dermatomes and flaccid weakness results in muscles which receive innervation from the injured segments. The tendon reflexes are affected accordingly. They are abolished or diminished in the damaged segments and exaggerated in the segments caudad to the injury. This is a good diagnostic guide to the level of the lesion.

Increased and spontaneous reflex activity occurs distal to the affection even in incomplete lesions of the long tracts (SCARFF). The positive Babinski toe sign does not follow all transections of the cord.

There are different disturbances in the function of the autonomous nervous system depending on the level of neural destruction. It is of importance to note that affections of abdomen and pelvis are painless if the lesion is above the sixth dorsal segment. Another important fact to be taken into consideration is that the blood pressure does not fall markedly and no true surgical shock ensues injuries of the spinal cord (SCARFF SMITH WHITE). If a paraplegic patient is in state of shock, the cause of it should

be sought for. There must be some associated injury which deserves attention and which is responsible for the fall of blood pressure.

Injuries to the cauda equina produce typical root symptoms with sensory changes, reflex disturbances, flaccid paralysis or paresis with possible fascicular twitchings. Pain, hyperaesthesia and hyperalgesia are common symptoms. That is not the case in lesions of the spinal cord.

Bladder, bowel and sexual functions are almost invariably affected in all injuries of the spinal cord or cauda equina. The mechanism of micturition is complicated. Stretching of the bladder provides the normal stimulus for emptying. Higher centers inhibit the emptying reflex until the act of micturition is initiated. Neurogenic bladder dysfunction can be of various types. Boshes divides them to uninhibited, reflex, autonomous and atonic bladders.

Uninhibited bladder is closest to normal and is seen in less severe or complete lesions. Normal voluntary control is possible, there is urgency with little frequency, but no residual. Reflex bladder results when the upper motor neurone is interrupted but the reflex arc is intact. The patient voids precipitously and there is generally a residual left in the bladder. Autonomous bladder is without external innervation. Voiding is affected by increased intra-abdominal pressure and is never complete. This is seen in some injuries of the conus medullaris and cauda equina. In atonic bladder there is no sensation and no desire to void. That is the situation during spinal shock, for example.

Bowel paralysis subsides fairly rapidly and reflex evacuation of the rectum will be elicited by distension. The amount of sexual dysfunction is quite variable. The function of erection is a rather simple one and is possible even when the cauda equina is destroyed. Disturbances in sexual relations are common but some kind of gratification can be elicited if erection persists.

The roots of cauda equina are able to recover or regenerate like other peripheral nerves. Thus the injury can be neurapraxia, axonotmesis or neurotmesis, as well as a combination of these in character.

\*

### Complete or Incomplete Lesion of the Cord?

If the paraplegia is partial and segments distal to the site of injury show some function, it is clear that the cord lesion is incomplete. But total suppression of cord function below the level of injury can be due to anatomo-



mical transection of the cord or to *spinal concussion* in connection with an incomplete cord lesion. The immediate effect of cord division is the production of spinal shock. Now a question of practical importance arises. Is it possible to differentiate in the first few hours, spinal concussion from spinal shock? If we can be sure that the patient is in the state of spinal shock then no operation need to be contemplated. But if there is evidence of spinal concussion we have to decide quickly which would be the best treatment to provide maximum recovery. HOLDSWORTH and HANAU state that *spinal concussion* is distinguishable from spinal shock within 12 hours after injury. Repeated meticulous neurological examination would show some recovery from concussion very early. First comes sensibility then motor power and later reflexes. Reflex activity never comes first in spinal concussion. On the contrary recovery from spinal shock is marked by the return of exaggerated segmental reflexes change from flaccid to spastic paralysis but no recovery of normal sensation or normal motor power (HOLDSWORTH and HANAU). Some authors regard that recovery from concussion can start later but will occur in 48 to 72 hours (BOULET, quoted by VIGOR). CUTTMANN, JEFFERSON quoted by HOLDSWORTH and VEFFERTEN. Other writers share the opinion that recovery can start still later (BUSHNICK, SCARFE and STANGER). VAFFERTEN describes a clinical test which he considers most practical and informative in distinguishing incompleteness from completeness of cord division. The examiner should forcibly flex one of the patient's toes and compress it in such a manner as to cause severe pain. If the patient perceives some sensation from this test the interruption of the cord is regarded as incomplete.

\*

### How to Distinguish Root Lesions from Cord Damage?

This is important because root lesions can recover through regeneration which can take a long time. The spinal cord terminates in a bulb at the distal end of the first lumbar vertebral body. Injuries distal to this are called pure root lesions. But higher up root lesions are to be distinguished from cord damage. In fracture dislocation of the spine the cord is damaged at the level of the vertebral injury (HOLDSWORTH). If this does not correlate with the segmental level of the paralysis the disparity is due to root lesion. Comparison of the clinical neurological level with the level of cord lesion determined by the radiograph will thus show the pattern of cord and root damage (HOLDSWORTH). The relationship of spinal cord and root lesions

vertebral bodies varies to some extent from patient to patient. According to HOLDSWORTH the first lumbar segment lies opposite the 9th dorsal vertebra so that between it and the first lumbar vertebra lie all the lumbar and sacral segments of the cord. Opposite the thoraco-lumbar vertebral junction lies the lumbosacral segmental border. According to BOSCH the first lumbar neurological segment is situated more distal opposite the disc between 10th and 11th dorsal vertebrae. Naturally this gives only a rough idea of the actual conditions. The spinal segments are not clearly defined, the anatomy is not exactly the same in all patients and the mechanism of injury varies. But it is possible to make valuable observations from repeated meticulous neurological examinations. When these are correlated to radiological findings, all necessary information is available.

\*

### Stable and Unstable Spinal Injuries

Classification of injuries into stable and unstable fractures is as sensible in the dorsolumbar spine as elsewhere in the body. The former are safe injuries which do not need special protection. The latter are unsafe lesions requiring support to prevent further damage. Reduction and fixation in order to maintain the reduced position is another problem and should be considered separately.

HOGLES regards as safe all vertebral body fractures which do not involve the posterior wall of the centrum, probably over 90 per cent of the cases. He includes to the unsafe group all dislocations and crush fractures involving the posterior wall of the centrum. NICOLL considers stable anterior and lateral wedge fractures of the vertebral body and all fractures of the laminae proximal to the fourth lumbar lamina. NICOLL holds as unstable all fracture subluxations with rupture of the interspinous ligament, all fracture dislocations and fractures of the laminae of the 1th and 5th lumbar vertebrae. He stresses further that most wedge fractures of severe degree should be regarded as unstable because they are in fact fracture subluxations with rupture of the interspinous ligament. WATSON JOYLS points out that the stability or instability of nearly all joint injuries and fracture dislocations is related closely to associated injuries of the neighbouring ligaments. WATSON JOYLS writes: "If the interspinous ligament is not ruptured and there is no severe injury to the ligaments of the apophysial joints or to the intervertebral disc and its annular ligament, fractures of the vertebral bodies are usually quite stable in the position of slight deformity produced at the moment

of injury and they are safe from increasing displacement. On the other hand if the interspinous ligament has been completely ruptured or avulsed and there is tearing of the ligaments of the apophyseal joints the vertebral fracture is not stable and it is not safe there may well be recurrent and increasing displacement with a threat of late compression of the cauda equina or spinal cord.

Surprisingly the extent of neural damage does not bear a constant relationship to the severity of skeletal injury. Stable fractures can produce total incurable paraplegia although this is rare and there can be unstable fractures with marked displacement but without neurological damage. This has been confirmed by many authors e.g. NICOLL, SEEVER, STANLEY, WATSON JONES.

Division into stable and unstable injuries does not have any practical purpose in the upper thoracic region perhaps above the 9th or 10th dorsal vertebra. Here the situation considers pure cord lesion nearly always complete and internal fixation with plates and screws would be useless and impractical because of weakness of the spinous processes. Furthermore the use of plaster jacket or plaster bed is contraindicated according to e.g. GUTTMANN, HOLDSWORTH and HARDY, NICOLL, WATSON JONES.

\*

### Operative Indications

Before commencing discussion of operative treatment it is important to make clear distinction between open and closed injuries. There is universal agreement that all open injuries should be operated on (GUTTMANN, HOLDSWORTH, WHITE and others). The lesion should be explored, foreign bodies must be removed, a meticulous debridement should be performed and antibiotics should be given. Open injuries are usually due to fire arms and explosions and are therefore rare in civilian life.

The treatment of closed injuries with paraplegia is still a matter of controversy. GUTTMANN who has vast experience of paraplegia writes: "In closed spinal injuries due to fractures or fracture dislocations this writer is in complete agreement with those who advocate conservative treatment and are strongly opposed to laminectomy as an immediate measure. This applies to injuries with complete transverse spinal syndromes at any level including cauda equina lesions. It may be emphasised that at this stage the Queckenstedt test is of no diagnostic value in differentiating subarachnoid block.

caused by oedema of the injured spinal cord or pressure from the dislocated bone\*

As to the incomplete paraplegias due to cord or cauda equina damage he is almost equally conservative. Nevertheless, GUTTMANN is not totally negative towards operation. He sees indications for laminectomy during the early stages in the following conditions. Incomplete lesions which show signs of progression, persistent manometric block without evidence of fracture or fracture dislocation in the vertebral column, regardless of whether the spinal syndrome is incomplete or complete. Irritation to nerve roots of severe degree caused by fragments of bone or disc prolapse is a further indication to laminectomy, although a very rare one. These lines are followed also by HOLDSWORTH, PINNABACKER and WATSON JONES.

Some surgeons are clearly in favour of early laminectomy in most cases (HAGLSTAM, STRLET, FARLOW, WANNAMAKER). Others, like COMARR, LOYAL DAVIS and FRYKHOLM regard it indicated mainly or only in the presence of demonstrable block in the spinal subarachnoid space. But according to HOLDSWORTH laminectomy is rarely of value in such patients for the maximum neurological damage is sustained at the time of injury and if the cord is not divided recovery will occur without decompression. He writes further: "In partial cord lesions with anatomical displacement of the spine restoration of vertebral alignment is sufficient to free the cord from pressure whilst fixation will prevent further damage."

Open reduction with internal fixation or to be more accurate internal splinting performed after the reduction is a rather new approach to the problem (HOLDSWORTH and HARDY 1953). Operative replacement is regarded imperative in fracture dislocations with locking of articular processes and desirable in fractures of the vertebral body involving the posterior wall of the centrum (ROGERS, WATSON JONES). When open reduction is performed it would seem advisable to proceed with internal fixation. With the exception of the conditions mentioned above the value of reduction is questionable. WATSON JONES considers reduction desirable in most cases whilst STANTON doubts whether it matters at all. The method of internal fixation with plates and screws or bolts is favoured by BARNES, DICK, HOLDSWORTH and HARDY, MURLE d'ARIGNI and BLANSSY, NICOLL, WATSON JONES, WILIS and WILLIAMS among others. This applies to certain fractures and fracture dislocations of the dorso lumbar spine. The main endeavour of it is to protect nerve roots and sometimes the incompleteness of cord lesion from further damage. If this policy can save some paraplegics from wheel chair life it has certainly served its purpose and proved its justification.

Furthermore it may make the patient more comfortable in the early stages of nursing

As to the stable fractures where no protection is needed apart from good nursing of course the indications for operation might well be those stated by GUTTMANN

The value of late laminectomy is worth considering GUTTMANN states as a general rule that it does not serve any useful therapeutic purpose in complete transverse lesions at any level But on the other hand it is indicated in incomplete lesions which show increasing neurological signs The late progression can be caused by callus or scar formation WHITE writes 'Late laminectomy is of doubtful value though it is occasionally helpful in relieving radicular pain'

*The treatment of intractable pain and violent spasms etc is out of the scope of the present investigation*

## OWN INVESTIGATIONS

### Description of the Material

During the years 1952—1962 altogether 39 cases of traumatic paraplegia of the thoracolumbar spine were treated at the Clinic. In three of the cases the radiographs are lacking or the notes are insufficient for evaluation. The material thus consists of 36 patients: 30 males and 6 females. Most of the patients were young adults; there were neither children nor old aged people in the series. A few of the patients had got primary treatment elsewhere and were sent to us later. Due to the fact that there are no paraplegia centers in Finland yet, unfortunately almost all patients had to be transferred to various hospitals later for further care.

Of these 36 patients with traumatic paraplegia of the thoracolumbar spine, seven died shortly after the accident. One case is too recent for ultimate neurological evaluation. In all the other cases we have either information of several years duration or strong evidence that no recovery is likely.

TABLE I — *Causes of fractures*

|  |    |
|--|----|
| Fall from height                               | 18 |
| Crushed  | 8  |
| Traffic accidents                              | 5  |
| Hit by falling object                          | 4  |
| Fall on the ground while carrying heavy weight | 1  |
| Total cases                                    | 36 |

Table I shows that half of the injuries were due to fall from height. Most patients fell from high scaffolding while at work; some fell from the roof by slipping; three attempted suicide by jumping down from a window and one came down 26 metres in a falling crane. The mechanism of crushing took place in various ways: collapsing structures, crushing under heavy slow vehicles etc. From the five traffic accidents four were due to motor

bicycles and one was a car collision. Hit by falling object was usually due to a rather heavy weight which the patient partly escaped from.

Distribution of fractures among various vertebrae is seen in table II.

TABLE II — *Distribution of the fractures among different vertebrae*

|                           |      |             |
|---------------------------|------|-------------|
| Thoracic                  | I    | —           |
| "                         | II   | 1 fracture  |
|                           | III  | —           |
|                           | IV   | 1 fracture  |
|                           | V    | 3 fractures |
|                           | VI   | 3 "         |
|                           | VII  | 3 "         |
|                           | VIII | 1 fracture  |
|                           | IX   | 2 fractures |
|                           | X    | 4 "         |
|                           | XI   | 4           |
|                           | XII  | 10 "        |
| Lumbar                    | I    | 9           |
|                           | II   | 3           |
|                           | III  | 2 "         |
|                           | IV   | 1 fracture  |
| "                         | V    | —           |
| Total fractured vertebrae |      | 47          |

The tendency of injuries to affect particularly the thoraco-lumbar junction is clearly seen even in this small series. This fact is well presented in the relevant literature. Before analysing the material closer we feel justified to divide it into three different groups. The first of these contains fractures of the dorsal spine proper, namely fractures of D1 to D11 inclusive. In the second group are fractures of the dorso-lumbar junction, that is D12 and L1. The third group, fractures of L2 to L5 consist those paraplegias only which affect the cauda equina.

## Fractures between 1st and 11th Dorsal Vertebrae — Practically Pure Cord Lesions

The 14 patients in this group had 22 fractured vertebral bodies. In three cases were three vertebrae fractured simultaneously and in two cases were two fractured vertebral bodies. There were four deaths in this group. All of them had multiple fractures and other injuries which directly or indirectly contributed to the patients death. The cause of death was twice cerebral contusion, once subarachnoid hemorrhage and severe tracheobronchitis with atelectasis of the lungs in one case. The latter had fracture of the second dorsal vertebra with high paraplegia. Seven of the ten cases left showed no neurological recovery. In three instances some improvement was noticed. In two of these cases the improvement was slight and in one case rather marked. These were fractures of D 10 and D 11. The two with slight improvement were treated by immediate postural reduction and plaster jacket. The plaster jacket had to be taken off fairly soon because of complications. One of the patients developed pressure sores under the plaster and the other got abdominal distress. Both patients were then treated in a plaster bed for a while and subsequently in ordinary bed rest. The complications cleared off gradually, the postural reduction was not fully maintained but some neurological recovery could be traced. The sensibility impaired somewhat and the proximal muscles of both lower limbs got some power. This allows walking with crutches although it is difficult partly because of weakness and partly because of spasticity.

The one particular case which showed marked improvement has some interesting features and will be presented in detail as follows.

26 years old laborer was involved in a tractor accident. He got jammed under the vehicle and incurred severe pain and deformity in the back. He could not move his lower limbs and told that he was not aware of the lower part of the body for a while. The pain was felt only in the back although he had compound fracture of the left tibia. The patient was immediately transported to the local hospital. He did not sustain surgical shock, was conscious all the time and complained of aching pain in the back and lower abdomen. Clinical examination was reported to have shown complete paraplegia below the level of the lesion. The radiograph disclosed unstable frac





Fig. 1



Fig. 2

Fig. 1 — Fracture dislocation of the 10th dorsal vertebra with locking of the articular processes. The injury produced immediate complete paraplegia below the level of the lesion.

Fig. 2 — Open reduction and stabilisation with plates and screws has been performed.

ture and dislocation of D 10—D11 with locking of articular processes (fig. 1). Twenty-one hours after the accident the patient was transferred to us. Neurological examination revealed now hyperesthetic dermatomes D11—D12 and the dermatomes L 1 and L 2 were not anesthetic any more they were hypesthetic. Below that level there was complete anesthesia. All myotomes below the level of the injury were paralysed. Only very weak anal reflex could be traced.

The question arose how to act in this case. Twenty-one hours after the injury the anal reflex was weakly present. dermatomes D11 and D12 were hyperesthetic. dermatomes L1—L2 showed some recovery and that was all positive in otherwise complete paraplegia below the bony lesion. Should these signs be regarded as proof from completeness or incompleteness of the cord lesion? Dermatomes L1—L2 could belong to the distal end of the proximal cord segment and the signs could mean beginning disappearance of spinal shock. But if this were incomplete cord lesion with locking of the articular processes how could the cord be protected? We regarded operation open reduction and internal fixation with plates and bolts justified.



*Fig 3 — The plates could not prevent gradual slipping of the fracture during three months but this did not disturb good neurological recovery*



*Fig 4 A*



*Fig 4 B*

*Fig 4 A and 4 B — Position of the fracture 19 months after the original injury. The neurological recovery remained good and there was no deterioration of function later. The plates were removed four months after their introduction.*

The operation was performed under general anesthesia reduction was achieved by excision of one of the facets and the fracture dislocation was stabilised by Williams plates and screws (fig 2) The lamina of 11th dorsal vertebra was fractured and loose and had to be removed before the plates were fixed

The following day we could notice that dermatomes L3 and L4 were working 1 week after the right hamstring muscles contracted voluntarily and so did extensor digitorum longus in the left foot Three weeks after the operation the patient could move his right foot and contract voluntarily muscles in both thighs the knee jerks could be traced as well as the right ankle jerk The patient could urinate and pass faeces without help the indwelling catheter drainage was removed Sensibility was normal except in a small area distal to the skin wound in the left leg The plates could not prevent gradual slipping of the fracture during three months (fig 3) but this did not disturb good neurological recovery

The follow up examination 19 months later showed that the patient could walk without any help but preferred to use on stick outdoors The patient had uninhibited bladder function evacuated normally had useful erection but no ejaculation Sweating in the lower limbs was exaggerated There was some spasticity in the legs too but this seldom caused trouble All qualities of voluntary movement were working although there was weakness in certain groups of muscles specially in the left leg The plates had been removed four months after their introduction The patient did not complain of any pain He also had good movement in the back Radiographs showed that the position of fracture was nearly the same as before the reduction (fig 4 A and 4 B)

The present writer regards it very likely that internal fixation in this case has contributed to the neurological recovery The writer feels further that the nursing of the patient in a busy surgical ward had been well aided by the plate fixation during the first few weeks after the injury It seems evident that the incompleteness of the cord lesion in this case could not have been protected without operative intervention of this sort

### Fractures of Dorso-Lumbar Junction (D12 and L1) — Mixed Cord and Root Lesions

This group consists of 16 patients with 19 fractures. Three patients had both vertebrae fractured. Three patients died within two weeks of the injury. One of them had pulmonary embolism, another had rupture of the pleura with pulmonary complications, and the third patient died from perforated ileitis with peritonitis and pneumonia. Nine patients showed no neurological improvement, and in three cases the neural damage increased clearly and markedly during the conservative treatment. They did not improve later either. In two of these cases the paraplegia was practically produced by postural reduction in hyperextension.

In four instances some recovery was noticed. The S1 dermatomes were hypesthetic and the myotomes L5 and S1 were weak twice. The disturbance in sensibility disappeared and the motor components gained strength. The third patient had both dermatomes and myotomes of L5—S1—S2 affected and these recovered partially. The fourth case was a fracture-dislocation of D12—L1 where the third and fourth lumbar roots showed some improvement in the beginning but this recovery deteriorated later again. The patient can now walk with crutches but he needs one splint. He had mixed cord and root lesion where some root recovery took place. Late laminectomy was performed with doubtful value.

Open reduction and internal fixation was taken into clinical use rather late and after its introduction no fractures of D12 or L1 with paraplegia have occurred so far.



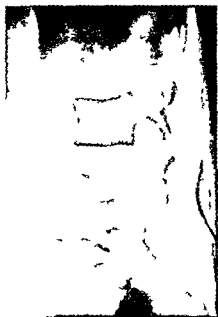


Fig. 5



Fig. 6

Fig. 5 — Unstable fracture of the third lumbar vertebra with paraplegia below the lesion. The patient complained of agonizing pain in the back and in both lower limbs.

Fig. 6 — Open reduction and internal fixation has been performed.



Fig. 7 A.

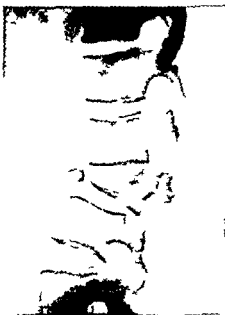


Fig. 7 B.

Fig. 7 A and B — The position of fracture deteriorated but marked neurological improvement continued. Control radiographs nearly a year after the injury. The plates were removed four months after their introduction.



Fig. 8A



Fig. 8B

Fig. 8A and 8B — Unstable fracture of the third lumbar vertebra with associated neurological signs. Complete motor paralysis developed gradually below L3 during the first 24 hours.



Fig. 9A

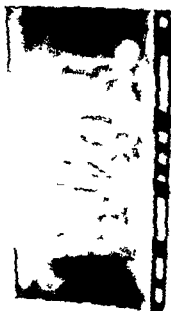


Fig. 9B

Fig. 9A and 9B — Open reduction of fracture was performed one day after the injury. The recovery started very well because remarkably good



Fig 10 A



Fig 10 B

Fig 10 A and 10 B — Fractures of the first and second lumbar vertebra with comminution of the corresponding laminae pedicles and spinous processes. There was partial paraplegia.

of L3 with paraplegia. At first he could move the right foot but gradually both feet became paralysed. He was immediately transferred to the nearest hospital and was sent to us the following day on admission there was a total motor paralysis below L3 the jerks were abolished but the dermatomes were not anesthetic. Dermatomes L3—S1 were hypesthetic on both sides and he had paresthesiae in both legs. The radiographs revealed unstable fracture of L3 (figures 8A and 8B). Open reduction and plating was immediately performed (figures 9 A and 9 B). Some improvement could be traced already the following day. Voluntary contraction in some muscles of the thighs could be noticed but no movement could be produced until several weeks. Normal urination became possible in a week. The sensibility recovered in two months and he could start walking with crutches three months after the injury. He returned to work as a laborer 8 months after the injury. The patient walks without support motor power mobility of the back and both lower limbs are about normal again. He gets some backache after heavy work but can manage with it. A new occupation is planned for him and vocational training starts soon.





Fig. 11 A.



Fig. 11 B

Fig 11 A and 11 B — A loose laminar fragment was removed and two long curved plates were fixed to five spinous processes. Reduction was not attempted because of gross comminution of the fractures and because of noteworthy function below the lesion. The symptoms were aggravated after the procedure.

The present writer feels that internal fixation played important role in the good neurological recovery of these two cases. The other three cases in this group of paraplegia did not show that amount of recovery. One of them was operated upon similarly and the other two were treated conservatively. The sixth case in this series is a recent one the observation period being not more than two months at the moment. But this case presents some special features of interest and is therefore reported here.

He is a 16 years old boy who was knocked down with his motor bicycle by a van. He sustained a partial paraplegia below L3. The radiographs disclosed fractures of L1 and L2, the latter being displaced. The laminae of both vertebrae were fractured and specially the lamina of L1 was severely comminuted and displaced (figures 10 A and 10 B). Hypesthesia below L3 and weakness in the motor power occurred below the level of the lesion. Because we did not have a Stryker frame it was difficult to decide should we operate immediately or not. Closed reduction and putting on a plaster jacket could be dangerous because of the instability with loose laminar fragments. He was transferred directly to the operating theatre where the

tion to the neurological state of the patient. The deterioration of reduction never increased neurological symptoms. This applies as well to the series of closed reduction with plaster jacket as to those of open reduction and internal fixation. The same observation was made by STAVORN in his series. Further aggravation of neurological picture was noted twice due to lack of fixation.

Eight of the patients got pressure sores and all had urinary infection at least in the early stages.

From the nine laminectomies three were done early. By early laminectomy we mean that it has been performed within three days from the injury. The other laminectomies were carried out a few weeks or several months later. No one of the early laminectomised patients showed any recovery. Half of the late laminectomies showed some recovery and half did not. Those who improved did so even before the operation and the improvement continued after the laminectomy. It is difficult to know the real value of laminectomy if there are no convincing objective signs of recovery because the patients usually are very anxious to see improvement and may therefore exaggerate their observations. The late recovery was always related to the nerve roots.

One of the patients treated by bed rest and one treated in plaster bed recovered slightly. All others which showed improvement had some form of «active» treatment. What is the real value of this «activity» is of course difficult to evaluate. One might state perhaps that they would have recovered anyway. On the other hand the only patients who showed marked neurological improvement had been openly reduced and internally fixed.

One must admit that all patients with internal fixation did not do well. One died in pulmonary embolism, two did not improve at all and one became worse. The latter case has been described in the previous chapter and we hope for ultimate recovery in that fairly recent case of cauda equina paraplegia.

Of the four favourable cases of internal fixation three recovered markedly and one improved slightly. It is to be noted that the position of reduction deteriorated even in this series towards the original situation in about three months. The plates could not resist weight bearing stress. Actually this is not surprising. The important fact is that the neurological recovery was not affected by the gradual sliding of the injured bones.

If open reduction and fixation with plates is of definite value in certain cases the question arises how does it work. One could think that reduction creates decompression in the injured area in the important early stage.

when hematoma and oedema are removed, fixation with plates protects the neural elements until the nature gets time to adapt to the circumstances. In many great deformities of the spine which develop gradually like scoliosis and Pott's disease neurological symptoms and signs are seldom seen.

In one of our cases where open reduction and fixation might have been beneficial we could not operate because of tuberculosis in the back. No operation was performed for fractures which we believed to have been stable. In one case of low partial paraplegia we did postural reduction and used plaster jacket for four months with moderate success.

The paralysed bladder was always treated with indwelling catheter drainage preferably of the Foley type and irrigated regularly. The catheter was clamped for some hours in order to educate the bladder to periodic reflex action. When this started to work the catheter was removed and the amount of residual urine was measured. If the residual remained negligible the indwelling catheter drainage was discontinued.

Summary of the fate of the present series of 36 paraplegics is as follows:  
7 patients died shortly after the injury.

20 of the cases showed no neurological recovery and in fact three of them became worse. In two instances the symptoms were grossly aggravated by postural reduction in hyperextension and one case deteriorated due to lack of support in the early stages.

In 9 paraplegics improvement of the neurological picture was noted. One of these deteriorated later again due to instability of the spine and one particular case worsened because all loose fragments were not detected at the operation.

In one patient only the recovery can be related to cord function in all other instances the improvement was due to root recovery. In the former and in two of the latter cases the improvement of neurological function was marked in the rest of the cases it was slight.

## DISCUSSION AND CONCLUSIONS

The fate of completely paraplegic patients is tragic. They are usually young people in their best years of life when the trauma to the spine occurred suddenly and unexpectedly. These patients fall easily into desparation and get passive if those responsible of their treatment and care can not encourage them by right approach to the problem. Traumatic paraplegia is a fairly rare condition. General and orthopaedic surgeons as well as other members of the hospital staff do not get experience in its treatment unless those patients are collected into special centers from the very beginning. This is fully realised in countries like Great Britain and the United States of America. Despite difficulties and psychological setbacks a great number of paraplegics have been rehabilitated to useful members of the society. They have been brought back to more or less active and even happy life. Visits to paraplegia centers such as Lodge Moore and Stoke Mandeville in England for example convince visitors of the remarkable results that can be obtained. Further experience will be gained and new hope is yielded to the forthcoming paraplegics that way.

The essential factors in the care of paraplegics are good nursing, prevention of complications and early rehabilitation. The surgical treatment of traumatic paraplegia seems still to be a matter of controversy. But as far as the present writer knows nobody claims that surgery would always be useless even in the closed injuries of the thoraco lumbar spine with paraplegia.

Hence we have to at least contemplate surgical aspects in evaluating the possibilities of neurological improvement in most cases. The patients or their relatives are anxious to know whether anything could be gained by surgical intervention. The surgeon takes heavy responsibility if he denies the patient a change, even a slight one. Surgeons responsible for the management of traumatic paraplegia must strain to clarify their indications of operation — and keep in mind that *prima regula est non nocere*. Both conservative and operative treatment can be harmful if the indications and contraindications are not properly considered beforehand.

In the present series the position of reduction was not fully maintained in any of the cases but this never aggravated the neurological picture. This applies as well to the closed reduction with plaster jacket as to the open reduction with internal fixation. The present writer's experience is in accord with that of NICOLL<sup>3</sup> and STANLIS<sup>4</sup> for example namely that the maintenance of the reduced position is not essential to good functional recovery. STANLIS goes as far as to state that he doubts whether reduction matters at all. The writer would not dare however to leave the displacement unreduced in cases of paraplegia. The reduction can provide decompression which might be decisive to recovery in some cases. If the surgeon can provide fixation to the bones the injured tissues could be protected during the critical first few weeks after the accident. If on the other hand a complete paraplegia produced by bony injury is above the 10th dorsal vertebral body one doubts the value of operative interference of any kind. Even closed reduction would be useless because it could not be retained. These cases should probably be treated by early rehabilitation only.

The writer has performed open reduction and internal fixation with plates and bolts eight times. In three of these cases marked neurological improvement was gained and it is likely that fixation has contributed to this since these are the only cases in the writer's series where striking recovery could be traced. The one case with incomplete cord lesion and spinal concussion which resulted to complete immediate paraplegia below the level of the lesion at as high as the 10th dorsal vertebra is particularly interesting. Now 19 months after the operation the patient walks outdoors with one stick and can e.g. voluntarily forcibly extend and flex his toes. It is in accord to HOLDSWORTHY<sup>5</sup> and HARRIS<sup>6</sup> teaching that spinal concussion and spinal shock should be and can be differentiated at the early stage of paraplegia. This case showed remarkable recovery of cord function and is probably rather unique.

The present writer believes that in his eight internal fixations the operation has been beneficial in four, useless in three and harmful in one of the cases. The latter teaches that good radiographs are mandatory for the evaluation of the bony injury and that the spinal canal should be explored if there is any doubt of the circumstances at the site of the lesion before the plates are fixed to the spinous processes.

It is to be noted that the paraplegia was practically produced by postural reduction in hyperextension and that in further two cases the neurological symptoms were aggravated during conservative treatment.

In the following we will summarize the main points of treatment.

and operative treatment at the early stage of traumatic paraplegia due to closed injuries of the thoraco lumbar spine. We may divide the early treatment into five categories: nursing in bed only, closed reduction and plaster jacket, open reduction and internal fixation with plates and bolts, nursing on a Stryker frame, and laminectomy.

*Nursing in Bed Only* — This applies without doubt to all fractures of the upper thoracic spine, perhaps above the 10th thoracic vertebra. The paraplegia at that level is usually complete due to division of the cord (HARDY). Nothing could be gained from surgery if there is definite bony injury. Massive disc prolapse, which is probably a very rare possibility, should of course be removed wherever it produces severe neurological symptoms. If we can regard the fracture stable, there is no need for fixation (HOLDSWORTH and NICOLL). The question of decompression arises. Some sort of postural reduction takes place anyway by nursing in bed as compared to the position at the moment of the accident. It is likely that no further decompression is necessary (HOLDSWORTH). GUTTMAN treats practically all his cases this way.

*Closed Reduction and Plaster Jacket* — If not contraindicated, this could be the treatment to some patients with low paraplegia if the fracture is relatively stable. The reduction would take place in hyperextension and the aim of plaster fixation would be to retain the reduced position. It is understood that the paraplegia should be below the first lumbar segment in these cases, otherwise pressure sores would inevitable occur under the plaster. For the same reason plaster bed is contraindicated (GUTTMAN, HOLDSWORTH and HARDY, WATSON-JONES and others). Contraindications for closed reduction in hyperextension are fracture dislocations with locking of articular processes, fractures of the vertebral body involving the posterior wall of the centrum (ROGERS and WATSON-JONES) and probably comminution of the laminae or pedicles.

*Open Reduction and Internal Fixation (or Splinting) with Plates and Bolts or Screws* — This is at present advocated by many authors in unstable fractures of the dorsolumbar spine (BARNES, DICK, HOLDSWORTH and HARDY, MERLE D'ALBIGNÉ and BENASSY, NICOLL, WATSON-JONES, WILES and WILLIAMS). If exploration of the fracture and laminectomy would be indicated for some reason, e.g. pressure by displaced fragments of bone, it would seem advisable to proceed with internal fixation. According to ROGERS, local anesthesia is preferable in open operative reduction.

*Nursing on a Stryker Frame* — An alternative to internal fixation is the use of a frame which permits turning of the paraplegic patient without for

sional strain on the spine (HOLDSWORTH) One would think that plating provides better support against danger of displacement if the spinous processes laminae and pedicles are intact A frame of the Straker type remains the only choice in grossly comminuted fractures or when plating cannot provide fixation (WILLIAMS)

*Laminectomy* — GUTTMANN and HOLDSWORTH believe that early laminectomy is indicated if progressive deepening of the paraplegia occurs in the hospital Increasing neurological signs during the transportation from the place of accident to the hospital can be due to the instability of the spine The treatment should then be directed towards stability If no fracture or dislocation can be detected in injuries of the spinal cord and nerve roots the QUECKENSTEDT test should be performed and in case of manometric block laminectomy should be done immediately regardless of completeness or incompleteness of the paraplegia (GUTTMANN) In late laminectomy the indications for operation can be chosen more freely When stability is achieved and the neurological behaviour of the patient is known we can estimate more accurately the possible gain or loss from a laminectomy The results of late laminectomy are contradictory Many surgeons in America are in favour of laminectomy both early and late but most British authors take a reserved attitude in the matter (BARRELL COMARU LOYAL DAVIS GUTTMANN HOLDSWORTH ROGERS SMITH TAYLOR WATSON JONES WHITE and others) HAGELSTAM of Finland published forty eight late laminectomies with fifteen favourable results Since late laminectomy contrary to the early one does not cause noteworthy instability it can not have the same contraindications than the latter It can therefore be performed with less restriction if there exist a slight change of improvement

It should be understood that the indications for operation discussed above stand for closed injuries of the thoraco lumbar spine with paraplegia All authors agree that immediate operative treatment is mandatory in every open lesion Those should be explored foreign bodies removed and meticulous debridement should be performed

In concluding the present writer would say as follows — Each new case of traumatic paraplegia is like an arithmetical example to calculate It is our first duty to make a careful clinical and neurological examination immediately take good radiographs and repeat the neurological examination in a few hours to trace any improvement or deterioration which might have taken place Then we have to correlate the x ray findings to the clinical picture After some hours of observation we should be able to evaluate the lesion and make a rough estimation of the prognosis





